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BRITISH MEDICAL JOURNAL

JOURNAL OF THE BRITISH MEDICAL ASSOCIATION

SATURDAY MAY 15 1943

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BRITISH MEDICAL JOURNAL

LONDON SATURDAY MAY 15 1943

HEAD INJURIES IN MOTOR-CYCLISTS

WITH SPECIAL REFERENCE TO CRASH HELMETS

BY

HUGH CAIRNS, D.M., F.R.C.S.

AND

H. HOLBOURN, D.Phil.

(From a Military Hospital for Head Injuries and the Nuffield Department of Surgery, Oxford)

In an earlier paper (Cairns, 1941) attention was called to the use of crash helmets to alleviate the head injuries of motor-cyclists, and 7 cases were reported. We now have records of 106 accidents in which we have been able to examine the crash helmet and, usually, the patient. This material provides further information about head injuries in motor-cyclists, and also enables us to compare the relative values of different types of helmet.

Two main varieties of helmet are in use. In one the outer shell is composed mainly of hard vulcanized rubber; in the other, of compressed wood pulp. Apart from their composition, there are important differences of construction. In the vulcanized type (Fig. 1) the inner suspension system is connected to the outer shell by means of a stout cord at the base of the helmet; if the cord is broken there is nothing to prevent the rider's head from coming into contact with the crown of the outer shell. In the pulp type (Fig. 2) the inner slings are stitched separately into the substance of the outer shell, and the stout cord at the base merely retains the hatband in place.

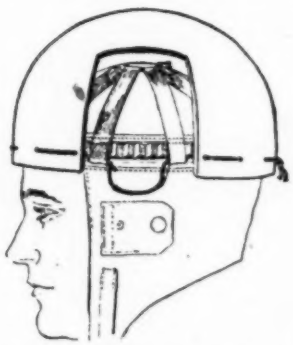

FIG. 1.—Vulcanized rubber helmet.

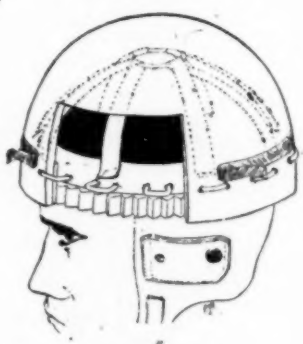

FIG. 2.—Pulp helmet.

Material

Our material consists of cases admitted to a Military Hospital for Head Injuries and extramural cases brought to our notice from various sources because of our interest in crash helmets (Table I). Of the hospital cases, those admitted after the acute stage were sent for assessment of their fitness to return to duty, for convalescence, or for special treatment; they were thus a specially selected group. The cases admitted in the acute stage were unselected, except that they did not include the accidents in which the motor-cyclist died before he could be moved to hospital, nor the mild accidents in which the motor-cyclist did not require hospital treatment. The immediately fatal cases and a few of the mild ones are represented in the extramural group—the fatal cases to a disproportionate extent. These have been largely traced through the help of ordnance depots receiving damaged helmets.

TABLE I.—*Head Injuries in Motor-cyclists With and Without a Crash Helmet (Figures for Non-crash-helmet Cases in Black Type)*

	Hospital Cases				Extra-mural Cases	Totals
	Admitted in Acute Stage		Admitted in Subacute or Chronic Stage			
Very mild (no amnesia)	1	(3)	3	(10)	9	13*
Mild (amnesia < 1 hour)	6	(6)	14	(21)	7	27*
Moderately severe (amnesia 1–24 hours) ..	8	(10)	23	(53)	4	35
Severe (amnesia 1–7 days)	4	(2)	7	(52)	1	12
Very severe (amnesia > 7 days)	2	(9)	3	(35)	1	6
Unclassified (duration of amnesia unknown) ..	—	—	—	(4)	3	3
Fatal	1	(3)	—	—	9	10
Totals	22	(33)	50	(175)	34†	106

* Six of the cases in these two groups were so slightly injured as not to require hospital treatment.

† In 2 cases a patient had two separate accidents while wearing the same helmet.

In classifying the cases according to their severity we have used the total duration of amnesia. With certain limitations common to all the subgroups, this provides a rough measure of the intensity of concussion. The severity of head injuries depends on other effects besides concussion—effects such as bruising, infarction, or laceration of parts of the brain, and intracranial haemorrhage or infection—but the clinical state of concussion, as judged by the duration of amnesia, is the only available measure.

In the first six case reports which follow we have attempted to give a fair sample of our material. Cases have been selected from the groups in Table I, so far as possible in proportion to the number of cases in each group. However, we have not taken a case from the "severe" group, since its cases closely resemble those of the "moderately severe" group.

A Very Mild Case

Case 1.—While travelling at high speed Cpl. A., aged 22, hit a rut in the road and was thrown on to his head. The left frontal region of his crash helmet (vulcanized type) showed a large area of abrasion and a fracture, and his triplex goggles were broken

into many fragments, but these did not become detached (Fig. 3). He had a cut in his left eyebrow, and his forehead was bruised. He felt dazed for some hours, but did not lose consciousness. He complained of headaches for four days, but remained with his unit. Six months later he was performing full duty efficiently. (Case of Surg. Lieut. B. Robinson, R.N.V.R.)

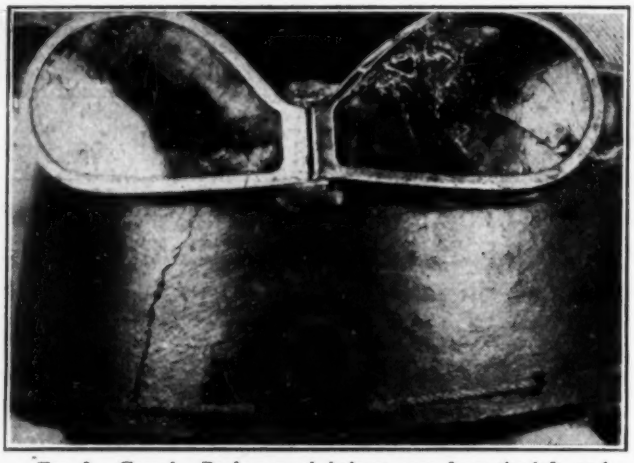

FIG. 3.—Case 1. Broken crash helmet, seen from the left, and goggles, which were worn upside down. The wearer was able to remain with his unit.

A Mild Case

Case 2.—Rifleman B., aged 19, was admitted to hospital after the second of two accidents at an interval of five days. In the first he had had a tyre burst while travelling at 30 to 35 m.p.h., and had gone into a ditch and pitched over the handle-bars. He woke up two minutes later and found himself sitting in the ditch. He was taken back to his unit in a lorry. Five days later, on his next cycle journey, he collided with a lorry which suddenly turned right when he was overtaking it on a main road at 35 m.p.h. This time he was amnesic for 10 minutes, and had bruises on the left side of his forehead, left shoulder, and both knees. Radiographs showed no fractures. After 23 days in hospital he returned to his unit. Although of normal intelligence, he was irresponsible to a degree that probably rendered him unusually prone to accidents.

His crash helmet (pulp type) showed high up in the right frontal region areas covered with a thin layer of earth that were probably produced by the first accident. Lower down in the right frontal region was an extensive area of deeper abrasions and lacerations that was probably caused by the second accident.

Moderately Severe Cases

Case 3.—Cpl. C., aged 24, also had two accidents. In the first, which belonged to the very mild category, he ran into a car that was on the wrong side of the road. His right external malleolus was struck by the bumper, and his handle-bar caught the front-door

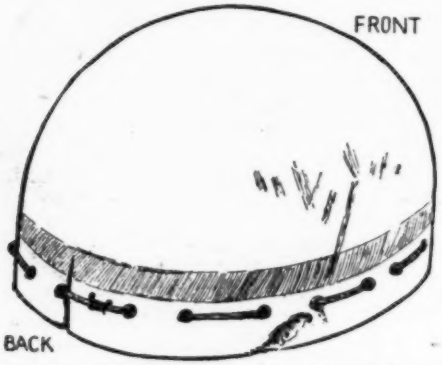

FIG. 4.—Case 3. Fracture at back of helmet (first accident). Indentation and abrasions on right side (second accident).

handle of the car. He was flung off on to the back of his head, but did not lose consciousness. He was unable to stand on his right leg for a quarter of an hour after the accident, but remained at duty and was riding a motor-cycle again next day. His pulp helmet showed a fracture in the occipital region (Fig. 4), which was identified by the patient and his commanding officer as being the result of this accident.

One month later he ran into a flock of sheep. This time his head injury was moderately severe: he was amnesic for two hours and had bleeding from the right ear, with a fracture of the right squamous temporal bone running into the petrous bone. His crash helmet (the same one) showed abrasions on the right side, with a dent at the edge (Fig. 4). He also had bruises of the right shoulder and elbow. He was kept in bed for 17 days, and then, after being up for only half a day, was transferred to a convalescent hospital, where he at once began to have headache and dizziness on exertion and soon became depressed. He was therefore eventually admitted to this hospital. Examination showed no signs other than the fracture of the skull. He was reassured, given graduated exercise, and returned to duty 12 weeks after the second accident.

These cases illustrate the value of crash helmets, and they also raise certain other aspects of accident prevention which, though beyond the scope of this paper, nevertheless deserve more attention than they have received in the past. In Cases 2 and 3 the successive accidents within a short period suggest that these patients might have been more prone to accidents than normal, and that after one accident the liability to accidents is for a time increased.

In Case 3 the treatment given after the second accident was not satisfactory. There is still a tendency to keep patients in bed too long after a mild head injury and then to discharge them from hospital without adequate reassurance or any attempt at physical hardening. In these circumstances unwonted exertion is often followed by headache and dizziness, and the patient, now beyond the control of a medical officer and perhaps at the mercy of over-sympathetic relations, loses confidence in his recovery. His fears aggravate his symptoms and may render him unfit for duty.

Case 4.—Signalman D., aged 28, had a head injury on Nov. 27, 1941. According to another dispatch rider, who was following him, they were travelling at 40 to 45 m.p.h. along an arterial road, when a car in front of them suddenly pulled out, and the patient had to brake so hard that he skidded and fell, striking his head against the kerb. His crash helmet (pulp type, Fig. 5) was penetrated in the

FIG. 5.—Case 4. Penetrating injury and abrasions of pulp helmet.

right frontal region; the surrounding area showed lines of fracture and numerous abrasions, deepest at the anterior edge of the helmet. He had a retrograde amnesia of 3 to 4 minutes and a post-traumatic amnesia of just under 24 hours. Radiographs of the skull showed multiple fissured fractures of the right fronto-parietal region extending on to the greater wing of the sphenoid (Fig. 6), but without any depression. The right fronto-malar suture was also separated. When he was examined two months later no abnormality of the central nervous system could be detected. He did well, and returned to his unit 10 weeks after the accident.

In this case the blow must have been particularly severe, for helmets of this type rarely show gross fractures, and as a rule the skull beneath them is not fractured. It is likely

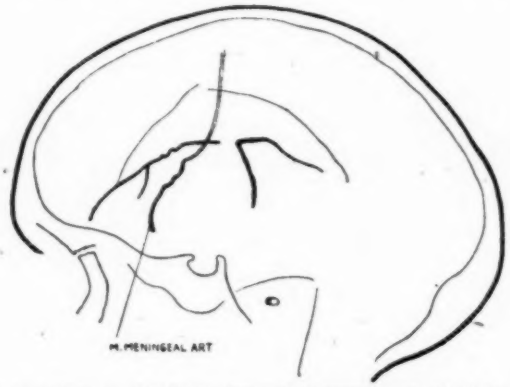

FIG. 6.—Case 4. Multiple fissured fragments of skull in right fronto-parietal region without depression.

that without the protection of a helmet this man would have had a compound depressed fracture of the skull, with penetration of the dura and severe, perhaps fatal, laceration of the brain.

A Very Severe Case

Case 5.—L'cpl. E., aged 24, ran into the back of an unlit stationary lorry in the middle of the night. He had a small scalp wound above the left ear, with a Y-shaped fracture of the underlying squamous temporal bone, and a superficial graze on the left cheek; he also bled from the right ear. His retrograde amnesia (estimated four months later) was 8 days and his post-traumatic amnesia about 20 days. On admission to hospital he was in coma, with spastic limbs, bilateral ankle clonus, and extensor plantar responses on each side. Within 12 hours he became violently restless, and remained in this state for 8 days; but after hot baths he became quieter. He was, however, still confused for some days, and during this time he had an incomplete and temporary right facial palsy of the peripheral type. The signs of organic neurological damage cleared up completely, but he still complained of headaches on exertion and gross impairment of memory. Attempts at active physical and psychotherapeutic rehabilitation failed, and he was invalided from the Army 4½ months after the accident.

His crash helmet (pulp type) showed a fracture with slight depression at the left lateral edge. Beneath this fracture the rim of the fibre hatband was broken, and the break corresponded with the scalp wound above the left ear.

This patient had a severe brain injury, with fracture of the base of the skull, which might have been fatal if he had not been to a considerable extent protected by the crash helmet from the full violence of the blow. Eventually he made a good recovery; but after such a prolonged period of post-traumatic coma and amnesia the capacity for satisfactory work may be slow to return, and in this case recovery was complicated by the fact that before the accident his adjustment to Army life had not been altogether satisfactory.

Fatal Cases

Case 6.—L'cpl. F., aged 27, came over the crest of a hill to find an oncoming car turning right towards the entrance of a station. His cycle struck the offside front wheel of the car, and he was thrown a distance of 40 feet, striking his head against cobbles (Fig. 7). His crash helmet (vulcanized type) had an

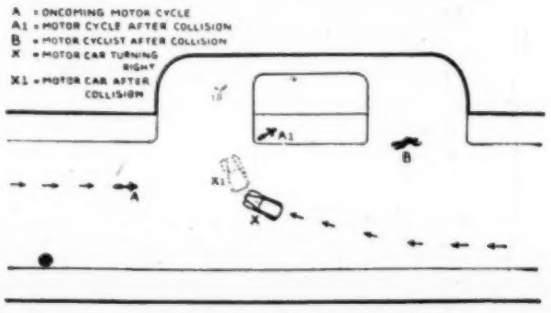

FIG. 7.—Case 6. Diagram of accident.

extensive fracture on the left side (Fig. 8), and there was an extensive fracture also in the left temporo-parietal region of the skull, spreading into the middle fossa. The patient never recovered consciousness, and died 51 hours after the accident. Before death he had right hemiparesis and rapid bubbly breathing. Necropsy showed, in addition to the skull fracture, extensive laceration and

FIG. 8.—Case 6. Fracture of vulcanized helmet.

haemorrhage of the left hemisphere beneath the site of the blow, together with a small amount of extradural clot. (From notes supplied by Brig. Hedley Whyte, D.S.O.)

In this case a less brittle and more strongly buffered helmet, protecting a wider area of the temporal region, might have prevented the fatal brain damage. Violent blows in the unprotected region of the external ear and mastoid process seem to be very liable to produce death.

In the fatal cases which we have examined death was not always due to head injury. In the following case—the only fatal case among those admitted to our hospital—the crash helmet was effective in preventing brain damage, but the patient died from bronchopneumonia secondary to injury of the thorax.

Case 7.—Travelling fast in daylight, with his head bent forward, Pte. G., aged 29, crashed into the back of a stationary lorry. Witnesses say he bounced back about six feet and landed sitting on his motor-cycle, then slowly keeled over sideways. A few hours later he was admitted to hospital in a comatose restless condition and showing signs of traumatic asphyxia. Temperature 103°; pulse rate 150; respiration rate 40. His crash helmet (pulp type, Fig. 9) showed

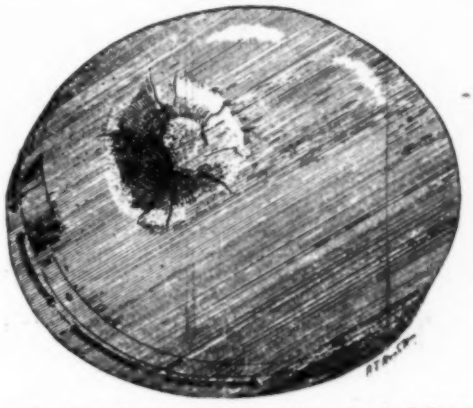

FIG. 9.—Case 7. Severe indentation of pulp helmet.

a large dent high up in the frontal region, with an eccentric deep semicircular mark which suggested that he had run into a piece of piping projecting from the back of the lorry, though it is stated that he hit the tailboard. There was also a deep transverse groove running across the posterior surface of the helmet. No signs of a lesion could be found in the scalp or skull. He had a bruise on the front of his sternum, fractures of upper thoracic vertebrae, and compound fractures of the left second and fourth metacarpals. Lumbar puncture (10 hours after injury) showed lightly blood-stained fluid under an initial pressure of 140 mm. H_2O, with a protein content of 100 mg. per 100 c.cm. and 7,200 red cells per c.mm. During the next three days he became less deeply unconscious; but his breathing

remained rapid and stridorous, and he gradually developed signs of bilateral bronchopneumonia, with a small pneumothorax on the right side, widening of the upper mediastinal shadow, disappearance of the heart sounds, and a persistently rapid pulse with gallop rhythm. He died four days after the accident.

Necropsy (carried out by Drs. Robb-Smith and Dorothy Russell) showed a fracture of the sternum beneath the bruise on the chest wall. There were considerable extravasation of blood in the upper mediastinum, slight bruising of the wall of the left ventricle of the heart, and bronchopneumonia. Neither the scalp nor the skull showed any sign of damage, and the only lesion found on the surface of the brain was a small amount of subpial haemorrhage along the medial sagittal border of each cerebral hemisphere. Coronal sections showed numerous petechial haemorrhages, with softening of the white matter, symmetrically scattered through both temporal lobes, linear haemorrhages in both fusiform gyri, and a few perivascular haemorrhages in the left frontal lobe. These were not sufficient to cause death, and on microscopical examination there were no signs of fat embolism. There was some bruising of the dura just above the foramen magnum and of the muscles of the neck. The spinal column showed a fracture-dislocation between the third and fourth thoracic vertebrae and a crush fracture of the body of the sixth thoracic vertebra, without injury to the spinal cord.

In this case there was undoubtedly a very severe blow to the head, and the crash helmet appears to have been most effective in preventing damage to the brain and skull; but it was not able to absorb all the energy of the blow on the head, and some of the force was transmitted to the spinal column. The primary cause of death was bronchopneumonia and mediastinal haemorrhage, due to a second blow on the chest and possibly also to the spinal injury.

Marks on the Helmet

From the marks on the outer shells of the helmets it is possible to deduce the positions of the blows and to some extent their severity. On the vulcanized type of helmet a serious blow generally causes a triradiate Y-shaped fracture of the outer shell, with its junction near the centre of the blow and the vertical limb usually extending to the edge of the helmet. With blows of still greater severity the upper arms of the Y may curve round and reach the edge, so that portions of the helmet become detached (Fig. 10).

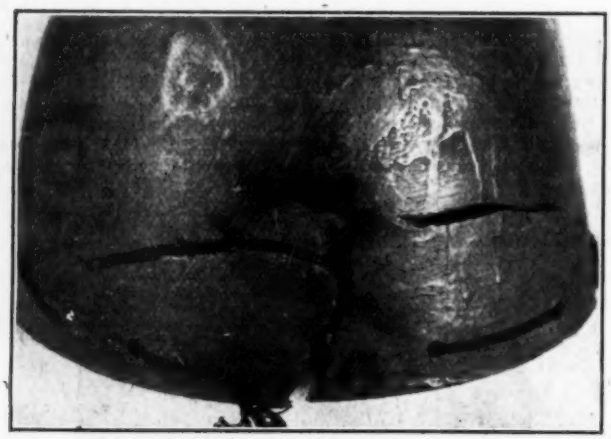

FIG. 10.—Vulcanized rubber helmet showing typical severe blow in the occipital region. The wearer was able to continue at duty.

The imprint of the object causing the blow is generally found near the junction of the Y. If the blow is due to the road inconspicuous groups of small pits, the imprints of the sharp corners of the road metal, are found at this point. The pits often have small tails to them, due to a small amount of "slip" between road and helmet. The pits may be easily overlooked, as they appear insignificant in comparison with the long and deep abrasions produced when the helmet is sliding along the surface of the road. And it must often happen that the pits due to the original blow are completely obliterated by abrasion marks due to subsequent sliding. In less serious blows the inconspicuous pits may occur without any fracture.

A blow by another vehicle usually causes a fracture. Near the centre of the fracture lines the paint may show an extremely faint polish at the summits of its irregularities, akin to the polish which occurs on the thicker parts of a starched collar from the pressure of the iron (Fig. 10). Sometimes the impact of another vehicle produces a smooth area denuded of paint.

The pulp type of helmet, being less brittle, seldom shows gross fractures; instead, an indented slightly softened area is produced by the blow. In this area there is slight wrinkling of the material, with little tendency for the wrinkles to run to the free edge (Fig. 9). Impact with the road usually causes small jagged tears of the layer of gossamer covering the helmet. The only clear evidence of impact with another vehicle is obtained in cases such as Case 7, in which an imprint of some geometric shape is seen in the helmet. No imprints of geometric shapes are found in the vulcanized helmets, presumably because the material breaks before it is sufficiently deformed to leave a permanent impression.

Multiple Blows

In 40% of 81 cases the helmet showed marks of more than one blow. In 2 cases the history and the character of the indentation in the helmet suggested that the patient struck two objects at once, one in the front part of the crown of the helmet and the other in the occipital region. In another case the helmet was crushed by the wheel of a lorry and fractured on both sides. In the remaining cases it is likely that the patients received successive blows against widely different areas of the helmet. There were indications in the marks that one blow was more severe than the others.

Case 8.—Pte. H., aged 27, struck the back of a lorry in daylight while riding his motor-cycle. He had a scalp wound in the forehead, a fractured frontal sinus, amnesia for two days, and double vision for two to three weeks. His crash helmet (vulcanized type) showed an irregular defect in the mid-frontal region, and, at the apex of this defect, depression of one fragment of the shell. In the right postero-lateral region the shell showed a second fracture, which was surrounded by areas of abrasion.

In this case there were clearly two blows—one on the front and one on the back of the helmet. Possibly the first blow was caused by the patient's head striking the lorry, and the second by his subsequent fall to the ground. That the main blow was on the forehead is shown by the severity of the damage to the front part of the helmet and by the fact that the damage to scalp and skull was beneath that area.

Evidence for multiple blows is also obtained from those cases in which there are fracture of the jaws and damage to the skull in some part other than the frontal and temporal regions.

Case 9.—Lieut. I., aged 24, crashed into a lorry which suddenly turned right into a side road. On admission to hospital two hours later he showed a simple fracture of the lower end of the left fibula, a comminuted fracture of the symphysis of the mandible, a fracture of the left condylar process of the mandible, and a fracture of the adjacent tympanic plate. There was no other fracture of the skull. His crash helmet was unmarked in the front, but showed a line of abrasions and a fracture in the left occipital region, with some general depression of the left occipital part of the helmet. The patient was amnesic for 7 days, and during the first part of this period was in a very confused state. He subsequently made a good recovery.

In this case there were obviously two blows—one on the face and one in the occipital region. Most of these cases of multiple blows appear to have occurred when the rider collided with a vehicle, such as a lorry or a taxi, and the first blow was probably due to striking the vehicle, the second to striking the ground. But in one fatal case of which we have read the report there is a circumstantial account that the rider fell and struck a fallen tree-trunk, then bounced up into the air, fell, and struck his head again.

Our observations confirm the experience of Rowbotham (1941) that "in road accidents in particular the head may be struck more than once." In the initial examination of a case of head injury, therefore, the medical officer should not be satisfied with finding one scalp wound, but should search the head for evidence of another. In a case which came to our notice neglect of this rule resulted in death from meningeal infection through an untreated wound of the hairy part of the scalp.

The Site of the Blow

The site of the marks on the helmet and the damage to the underlying scalp or skull showed a close correspondence. In 30% of our cases we could not establish a precise relationship—usually because there was no mark on the scalp or skull

(e.g., Case 7), but sometimes because the marks on the head, though close to the marks on the helmet, were not directly below them. In only 5% was there a definite variance between the site of the marks on the helmet and those on the head. In the remainder—i.e., in 93% of those in which requisite data were available—correspondence was precise.

The helmet thus provides a permanent record of the blow, and this is useful in patients who come under observation after their bruises and lacerations have healed. In some of our cases it has led to detection of fractures of the skull which had been overlooked at the first radiographic examination.

When the site of the main blow is plotted diagrammatically it is seen that blows on the frontal quadrant are almost as frequent as all the others put together (Fig. 11 and Table II).

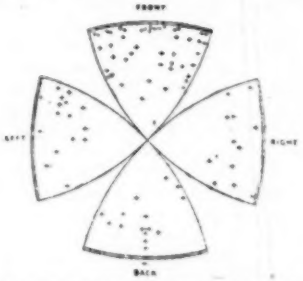

FIG. 11.—Diagram of site of main blow in 91 cases.

Blows on the front half of the helmet are over twice as frequent as blows on the back half. The majority are at or fairly near the edge of the helmet (the average height of blows is 4.5 cm. above the brim), and blows on the crown, an area approximately equal to that of a quadrant, are very rare: there were only 3 cases in which the blow was primarily on the crown. The

TABLE II.—*Effects of the Main Blow on Skull and Brain in Relation to Site of Injury**

Site of Injury	Total No. of Cases	Cases with Skull Fracture	Deaths attributable to Brain Injury
Frontal ..	47	16	3
Lateral ..	28	17	3
Occipital ..	21	3	—

case of the motor-cyclist is quite different from that of the equestrian, who starts his fall from a considerable height and has time enough to perform the rotation necessary for a fall on to the crown of his head. The motor-cyclist rides close to the ground, and in an accident usually falls from his machine in a forward, or lateral and forward, direction without enough height to perform the necessary rotation.

These observations show the importance in design of providing particularly good protection at and immediately above the edge of the helmet, especially at the front, where blows are two to three times as common as in any other part.

Effects of Blows in Different Regions

The outstanding feature of Table II is the mildness of the effects of blows in the occipital region in comparison with those on the frontal and lateral regions. It may be that in motor-cyclists a blow in the occipital region occurs only after the body of the motor-cyclist has almost come to rest; and that this is the explanation of the slightness of occipital injuries. Motor-cyclists probably do not turn somersaults in mid-air unless the initial violence is taken on some other part of the body before they turn over on to the occiput. Furthermore, the occipital region is more completely protected by the helmet, and below that by the neck muscles, than are other parts of the cranium.

Blows on the frontal and lateral regions are much more dangerous, and the figures of deaths and of fractures suggest that the lateral blow is the more dangerous of the two. The lateral regions are not well covered by the present designs of helmet, and the main effect of the blow may fall on the cranium below the level of the helmet. Similarly, the lower part of the frontal region is exposed to blows, particularly when motor-cyclists wear a helmet which is on the small side: in them the main violence may be applied to the unshielded supra-orbital ridge.

Of the 3 cases in which the blow was on the crown of the helmet one was the result of a mild glancing blow that produced only momentary amnesia. In the other two the blow was severe, and was associated with a crush fracture of the spine. One of these has been already described (Case 7); the other is as follows:

Case 10.—Driver J., aged 20, was following a van at 40 m.p.h. when it suddenly slowed and swung into the middle of the road in preparation for turning left. His cycle struck the near-side mud-guard of the now almost stationary van, and he was flung over the handle-bars. He was rendered momentarily unconscious, and had an abrasion on the bridge of his nose, pain in the back, and fractures of metacarpal bones of both hands. No signs developed in the central nervous system, but radiographs showed a crush fracture of the seventh thoracic vertebra. He returned to duty four months after the accident. (Major P. Clarkson's case.) His crash helmet (pulp type) showed numerous pitted and linear abrasions on the crown in an area 20 by 9 cm., extending forwards to the upper part of the frontal region. The patient's story was that his head had struck the back of the van; but this is unlikely, for in one of the abrasions was a fragment which was identified as road metal. In the inner sling of the helmet the stitches holding the two anterior loops to the outer shell were completely torn through.

It is significant that this case and Case 7 are the only two cases in the series in which a fracture of the vertebral column was found. However, the incidence of spinal column and spinal cord injury in motor-cycle accidents cannot be properly assessed without including a study of those patients who die soon after the accident and before they reach hospital; and in these cases thorough post-mortem examination is rarely made. In a series of 46 fatal motor-cycle accidents studied from the records of the Claims Section of one of the Army Commands there were three cases of instantaneous death ascribed, without necropsy, to broken neck. While the evidence is thus not very satisfactory, it does suggest that spinal injury is relatively uncommon after motor-cycle accidents, but that it should be looked for especially in those cases in which the blow is on the top of the head.

Absence of Marks on the Helmet

The value of head protection has been criticized on the grounds that brain damage is produced by violence applied to parts other than the head and transmitted to the base of the skull through the facial bones or the spinal column. In our series there were only 9 cases in which the helmet shell did not bear any marks of a blow, though the patient was regarded as a case of head injury.

Of these cases 5 proved to have had a head injury. In three of them there was laceration of one eyebrow below the crash helmet, which was too small for its wearer. In one case there was evidence of a blow on the leather flap covering the left ear. In the other case the lining of the helmet was broken and it is presumed that there was a blow on the head: this was one of our early cases, seen at a time when we were not yet expert at recognizing marks on the shell.

In 1 case the nasal bones were fractured and the patient did not become unconscious. In 2 cases the patient had a fracture of a limb bone, amnesia of short duration, and no evidence of a blow on the head or face. In these two cases it is possible that there was no brain injury, for amnesia may occur in uncomplicated cases of fracture of the femur in which there is no head injury. In one such case it was possible to demonstrate that, although the patient appeared to respond rationally during the stage of primary shock, he subsequently had amnesia for the greater part of the period. Another possible explanation for these cases is that there may have been a blow in the region of the foramen magnum transmitted through the vertebral column; but in the absence of any evidence of vertebral injury this seems unlikely.

In the ninth case, described as one of concussion, closer inquiry, after we had failed to find any marks of injury to the helmet, revealed that the man had not been concussed but had become amnesic from fright.

Case 11.—Gunner K., aged 27, was admitted to our hospital some two months after a motor-cycle accident suffering from the after-

* The number of cases was not large enough to enable us to estimate the relationship of the site of the blow to the duration of amnesia.

effects of head injury. The story obtained by the medical officer was that the man had collided with a car and that his retrograde amnesia was 1 to 2 seconds and his post-traumatic amnesia 6 hours. His complaints were of fairly continuous headaches, dizziness, and a feeling of weakness on attempting to do physical training. Examination showed no signs in the central nervous system. As his crash helmet bore no marks and he was obviously a rather nervous individual, his history was gone into more closely. Inquiry disclosed that there was in fact no retrograde amnesia and that his post-traumatic amnesia had not begun until half an hour after the injury, and had lasted until he was awakened 5½ hours later by a medical officer, who told him that he had had an accident. With the patient under pentothal, Capt. P. W. Nathan was later able to obtain a connected story of the whole period of amnesia and also fresh information about the man's anxieties and fears, which provided an adequate explanation of his symptoms.

It is probable that this case would have remained under a false label if absence of marks on the crash helmet had not led to closer inquiry.

Thus in the 106 accidents we are considering there is evidence of a blow on the head in all but 4. Of these four, one was a case of broken nose and another was a case of amnesia from fright. In the remaining two it is possible that slight brain injury may have resulted from violence applied to the base of the skull through the spinal column, though it is more likely that the brief amnesia which occurred was due to shock associated with fracture of a long bone. These observations do not disprove the contention that brain injury may result from injury to the face or spinal column, but they do establish the fact that in motor-cyclists brain injury is almost invariably associated with a blow on the head, protection against which can be provided by a properly fitting crash helmet.

Damage to the Helmet Lining

In considering the information which can be obtained from knowledge of the site of the blow we have dwelt of necessity on the damage to the outer shell, but it is important to recognize that the protective value of a helmet should rest to a great extent on its inner lining. With blows on the crown of the helmet the slings should absorb the energy of the blows, but they cease to do this as soon as the rider's head comes into contact with the outer shell. Owing to the rarity of blows on the crown, these slings are rarely brought into play; for in pulp helmets, in which it is to some extent possible to judge the strain placed upon them, the slings were broken in only two cases.

Against frontal, lateral, and occipital blows the hatband offers protection in proportion to its power to act as a buffer. This part of the inner lining could be altered with advantage, especially in the frontal region, where blows are most common.

The presence of a cord on the outer surface of the shell has been criticized by Gardner (1941) on the grounds that friction of the cords increases the deceleration of the head, thus rendering the effects of the blow more severe. We agree that the severity of the injury depends largely on the rate of deceleration, and that this rate can be reduced by sliding. Gardner suggested that the presence of the cord increased materially the risk of spinal injury. In our series of 104 cases there were only 2 cases of fractured spine, in one of which the blow was on the crown of the helmet and the cord was intact. We consider that the cord has proved to be an unnecessary feature of the design, and are thus in general agreement with Gardner. But we do not think that it is important in promoting deceleration, because the coefficient of friction between the surface of the helmet and the road, or whatever object the helmet strikes, is not altered adversely by the presence of the cord.

Our main objection to the cord concerns its use in the older type of vulcanized helmets as the connecting link between the inner lining and the shell (Fig. 1). In our series the outer cord was broken in 30 of the 52 vulcanized helmets and in only 9 of the 52 pulp helmets. This difference is significant, and is due to the fact that in the vulcanized type the cord is stretched when the helmet is struck and forced further on to the head; whereas in the pulp type the cord is not stretched as a result of the blow, since each sling, being stitched to the shell separately, is independent of the cord. As the helmet moves along the surface struck, the friction is usually enough to break the stretched cord of the vulcanized helmet, but does not suffice as a rule to break the unstretched cord of the pulp helmet. These effects may be compared with the well-known observation that when string is tightly stretched it can be much more easily cut with a pocket-knife than when it is not under tension.

The Theory of the Mode of Action of Crash Helmets

The chief functions of a crash helmet are two: (1) prevention of local injury in the region of the blow; and (2) prevention of effects remote from the region of the blow—viz., concussion, so-called contrecoup injury, etc.

Prevention of Local Injury.—The shell of the helmet acts partly by spreading the blow over a wider area, thereby diminishing its intensity at any one point. It is in this way, for example, that the helmet protects the scalp and skull from the pointed parts of the object struck, such as the sharp fragments of road metal projecting from the surface of the road. Hence, even by the action of the shell alone, some diminution in the number of fractures of the skull should be expected. In addition, some of the fractures should be prevented from becoming depressed, thereby reducing the liability to dural penetration and damage to the underlying brain. Since, owing to the stiffness of the shell, bending of the skull by the force of the blow is also lessened, there should be less contusion of the brain at the site of impact. The so-called "coup" lesion is, in our view, due to the bending of the skull under the blow.

The shell of the helmet also functions by "lengthening the blow"—that is, by spreading it out over a longer interval of time, so that it is not so intense at any particular instant. However produced, lengthening reduces not only the local injury but all other effects of the blow. The shell accomplishes this by sliding over objects for some time instead of stopping more abruptly, as the unprotected head would do owing to its greater coefficient of friction. But the main way in which the blow is spread over a longer time is by means of the buffering action of the slings and hatband. The blow lasts during all the time that the slings are being stretched or the hatband is being compressed, instead of only during the time taken to deform the scalp and skull. It is consequently reduced in intensity, and the local damage to scalp, skull, and brain is less severe. These parts of the existing types of helmet could be improved.

The blow is also lengthened to some extent by the rotation of the helmet relative to the head.

Prevention of the Remote Effects of the Blow.—The blow produces a change in the velocity of the head which may be either an acceleration or a retardation (Denny-Brown and Ritchie Russell, 1941). Calculation shows, however, that the change in the linear (or straight-line) velocity of the head produces only insignificant effects. The main effects are produced by a change in rotational velocity. As a rule the blow imparts a sudden rotation to the head. This is the cause of all gross bruises or lacerations of the cortex remote from the site of the blow. All the available evidence points to its being also the cause of the concussion. Anything which reduces the force of the blow will tend, other things being equal, to diminish the sudden rotation and its attendant lesions. Spreading the blow over a larger area is not effective from this point of view, since the total force is unaltered. But spreading it over a longer interval of time is to some extent effective, since the total force at any instant is reduced. Hence, owing to the sliding action of the shell and the buffering action of the slings and hatband, the helmet is effective in reducing the so-called contrecoup injuries and concussion.

Observations on the Effects of Crash Helmets

There are three groups of motor-cyclists to be considered: (1) those who die on the road before they can be moved to hospital—approximately 20% of 46 fatalities in one Army Command whose records we examined; (2) those who are

TABLE III.—*Influence of the Crash Helmet on the Incidence of Fracture of Skull and Duration of Amnesia (Hospital Cases only)*

	Acute Cases		Subacute and Chronic Cases	
	No Crash Helmet	Crash Helmet	No Crash Helmet	Crash Helmet
Fracture of Skull	63%	32%	39%	40%
Amnesia:				
Nil ..	3	1	10	3
< 1 hour ..	6	6	21	14
1–24 hours ..	10	8	53	23
1–7 days ..	2	4	52	7
> 7 days ..	9	2	35	3
Unclassified ..	3	1*	4	—
Totals ..	33	22	175	50

* Death due to causes other than head injury.

admitted to hospital; and (3) those whose injury is so slight that the motor-cyclist is not taken to, or is not detained in, hospital. Our information on the first and third groups is meagre. In the hospital cases there is a very significant difference between the motor-cyclists who were wearing crash helmets and those who were not (Table III).

Fractures.—Fractures of the skull are less frequent: as shown in Table III, of the patients admitted to our hospital in the acute stage only 32% of those wearing crash helmets had a fracture of the skull, as compared with 63% of those not wearing crash helmets. That the subacute and chronic cases do not confirm this finding is probably due, at least in part, to the fact that in those who wore a crash helmet our ability to find fractures long after the injury was considerably increased by knowing, from the marks on the helmet, where to look for them by means of further radiographic examinations.

Such fractures as do occur when a crash helmet is worn are probably much less severe than they would otherwise have been; for instance, the fracture may be fissured instead of depressed, as is perhaps exemplified in Case 4. In this hospital's series of injuries involving the frontal and ethmoidal sinuses, which have been collected by Major C. A. Calvert, there are 34 cases in which it is known whether or not a crash helmet was worn (Table IV). Among those who did not wear a crash

TABLE IV.—*Damage resulting from Fractures of Frontal and Ethmoidal Sinuses in Motor-cyclists*

	Without Crash Helmet	With Crash Helmet
Anosmia	14	1
Rhinorrhoea	7	—
Meningitis or brain abscess	2	—
Aerocele	1	—
Injury to carotid artery	2	—
Injury to optic nerve or chiasm	4	—
Displacement of eyeball or severe oculomotor paralysis	4	—
Disposal:		
Duty	12	8
Permanently unfit for military service	9	1*
Died	3	—
Under treatment	—	1
Totals	24	10

* Owing to malunited fracture of lower limb.

helmet severe fractures of the inner table of the skull are common: as the table shows, there are examples of cerebro-spinal rhinorrhoea, intracranial aerocele, meningitis, brain abscess, aneurysm of the internal carotid artery, lesions of the optic nerve or chiasm, and displacement of the orbit—in fact, most of the severe complications of extensively depressed fractures involving the frontal and ethmoidal sinuses. The patients who wore crash helmets were all free from these complications, with a corresponding improvement in the after-results. It is true that the chances of a patient being sent from a distance to this hospital are greater if he has one or more of the complications mentioned above than if he has none of them; but there is no selection as between those who have worn crash helmets and those who have not, and the most reasonable explanation of these figures is that the crash helmet is effective in preventing fractures of the frontal sinus from becoming depressed, from spreading widely, and from tearing the overlying dura, events which comprise one of the most important causes of morbidity and mortality among injured motor-cyclists. But to exercise this effect the helmet must come well down over the forehead, and, in view of the number of motor-cyclists who wear helmets which are too small for them, it is rather surprising that the results shown in Table III appear to be so uniformly favourable to the crash helmet.

Concussion.—In those patients who were wearing crash helmets, concussion, as measured by the duration of amnesia, was milder than in those who were not. As Table III shows, there is a significant reduction in the length of the amnesia: taking the acute, subacute, and chronic cases together, among the 208 cases in which no crash helmet was worn there were 44 (22%) with an amnesia of more than 7 days' duration; while among 72 cases in which a crash helmet was worn there were only 5 (7%) with an amnesia of more than 7 days.

Hence crash helmets cause a significant change in the percentage of fractures and long amnesias in those patients who come into hospital. The severity of injuries in a hospital population is not an easy thing to alter, and the fact that a detectable alteration has occurred implies that crash helmets have had a profound effect on the severity of injuries on the road. Unfortunately, it is not possible to give a mathematical "proof" that this alteration in severity has been in the direction of making the injuries milder, but common sense tells one that in most directions the change must have been for the better.

Some indirect quantitative evidence as to the effectiveness of crash helmets comes from a comparison between the two types of helmet. In the patients whom we have observed in hospital the distribution of the two types of helmet has been approximately equal (37 pulp and 35 vulcanized), but throughout the Army in this country during the period under investigation the issue of pulp and vulcanized helmets has been in the proportion of 2:1. The reasonable explanation of this is that half the accidents in wearers of pulp helmets were made so much milder than they would have been if a vulcanized helmet had been worn that the patient never came into hospital. The alternative explanation, that the pulp helmet made the accident so severe that half its wearers died before they got to our hospital, is rejected on the grounds that (*a*) the pulp helmet is the better helmet of the two (see below), and (*b*) such an enormous increase in the death rate would be easily seen from the figures of deaths to all motor-cyclists from all causes; these have, in fact, shown a decline since the introduction of crash helmets. No deductions can be made from this decline alone, since it is influenced by so many factors.

The estimate that the pulp helmet alleviates one-half of the injuries to such an extent that the dispatch rider does not require to go to hospital is a minimum estimate, based on the assumption that the vulcanized helmet had no effect in increasing or alleviating injury. But if, as we have reason to believe, the vulcanized helmet is of considerable use, this is an under-estimate of the effectiveness of crash helmets.

Using the minimum figure—that the pulp helmet saved one-half of its wearers from hospital—we can return to the data of Table III, which show that of the 72 hospital patients of all kinds wearing a crash helmet only 16 (22%) had an amnesia of one day or more, whereas of the 208 hospital patients not wearing a crash helmet 98 (47%) had an amnesia of one day or more. If we combine these facts with the supposition that there were at least another 37 pulp-crash-helmet accidents in which the riders never came to hospital because their injuries were so much reduced by their helmets, we obtain the result that the severe amnesias in 100 crash-helmet accidents number only about 15, at most, whereas in 100 non-crash-helmet accidents they number 47. We therefore deduce that the present helmets have reduced the severe amnesias to at least one-third of what they would have been.

Comparison of Types of Helmet

For a direct comparison of the two types we can use our figures for the total number of crash-helmet cases, which include many that did not come to this hospital and in which, since information was sought from all sources, there is a high proportion of fatalities. There were 52 of each type. Table V shows that in the vulcanized helmet group the outer shell is broken with much greater frequency, and skull fracture in cases coming into hospital is nearly twice as common. On the basis of these figures and those of the general distribution

TABLE V.—*Comparison of the Two Types of Helmet*

Type of Helmet	Total	Fracture of Helmet Shell	Fracture of Skull	Deaths	Remarks
Vulcanized	52	43	23	5	All due to head injury
Pulp	52	18	12	5	4 died of injuries to parts other than head

of the two types of helmet we may conclude that as regards prevention of fractures of the skull the pulp helmet is about four times as good as the vulcanized. If the vulcanized helmet were of no use in preventing fractures, then the pulp helmet would reduce fractures to one-quarter of their former incidence.

There is no significant difference in the duration of amnesias in the two groups of cases. But for this comparison our

figures are rather small. However, if we suppose that the serious amnesias in the two groups are in fact equal, then, since there are twice as many pulp helmets as vulcanized helmets in use, the serious amnesias per accident are twice as great in the vulcanized group as in the other.

Conclusions

In all, 106 examples of head injury in motor-cyclists wearing crash helmets have been examined.

The site of the blow on the helmet and the injury to the underlying scalp and skull correspond.

Over 50% of the blows are on the front of the helmet. The least common site of injury is the crown of the helmet.

Blows on the occipital region are least dangerous and those on the temporal region most dangerous to life. Blows on the crown may be associated with crush fractures of the vertebrae.

In 40% of the cases the head receives more than one blow.

In motor-cyclists it is very rare that brain injury results from a blow limited to the face.

The crash helmet is effective in diminishing local damage to the brain and its coverings at the site of impact, and it tends to lower the incidence of cases of prolonged amnesia.

Though our figures are rather small they suggest: (*a*) that the incidence of fractures of the skull is quartered by the better (pulp) type of helmet: the severity of those that do occur is less; (*b*) the incidence of prolonged amnesia (one day or more) is only one-third of that in accidents in which no crash helmet is worn; (*c*) in non-lethal accidents the pulp crash helmet so alleviates the injury that one-half of the dispatch riders who without its protection would have to go to hospital do not need to do so.

Of the two types of crash helmet in common use the pulp helmet is superior to the vulcanized rubber helmet.

Further improvements in the design of helmets offer a profitable field of preventive medicine.

We wish to acknowledge assistance from those officers mentioned in the text, also from Lieut.-Col. G. O. Chambers, *M.C.*, and the officers of a Military Hospital for Head Injuries—in particular Major W. Ritchie Russell; Brig. H. E. Rance, O.B.E. (Royal Signals); Col. E. R. Cawdron (Claims Commission); Col. F. P. Vidal, C.B.E. (Southern Command); Major C. Cassidy, *M.C.*; Major Patrick Clarkson, R.A.M.C.; Capt. Harold Elliott, R.C.A.M.C.; Sir Edward Penton (Chief Inspector of Clothing); Lieut.-Col. J. A. A. Pickard, D.S.O. (Royal Society for the Prevention of Accidents); and Mr. G. F. Rowbotham. The clinical notes on which this investigation was based were maintained by a grant from the Medical Research Council.

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A practical example of rehabilitation was mentioned at the sixty-third annual meeting of the Mental After-Care Association. The superintendent of one of the homes for convalescent male patients arranged with a local factory doing war work of much importance, first to train him in some semi-skilled processes with light and portable material, and then to send such material to the home in order that he might teach the patients to work upon it. Although this experiment was started only towards the close of last year, already the finished work sent back to the factory by the patients has greatly exceeded the firm's expectations alike in its accuracy and in the speed with which it has been done. Even more gratifying is the beneficial effect apparent in the patients. Their conversation at table is no longer about nerves and insomnia, and the reflection that they are taking some active part in the national effort is a great stimulus to their conversation, self-respect, and mental health. Col. Henry Yellowlees, chairman of the association, said at the meeting that the original object with which the association was founded was curative work, and the council had aimed at turning ex-patients into useful citizens, constructing a bridge over which the patients might pass from the restraints and seclusion of mental treatment to the freedom of responsible working life. Mr. R. Sargood, chairman of the Mental Hospitals Committee of the L.C.C., said that his council had no intention of doing this work itself, but was content to leave it in the hands of the association. The Bishop of London also supported the objects of the association (of which 13 bishops are vice-presidents), and spoke of the function of the pastoral office in helping people to regain mental health. During last year the association dealt with 2,500 patients, of whom over 1,000 were sent to the homes.

THE DIET IN DIABETES

BY

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Recent correspondence in the *Journal* has shown that some physicians are doubtful about the necessity for accurate dietetic control in the treatment of diabetes; it follows that they must be prepared to doubt the importance of maintaining the diabetic's blood sugar at a level near to normal. To all those who share this scepticism and to all those who are interested in the study of their less docile diabetic patients I offer, in full consciousness of all that I have still to learn, this short summary of my experience, conclusions, and practice.

Early in my experience as a physician, nearly 20 years ago, I found that it was possible to maintain excellent health and vigour in those rebellious severe diabetics who refused to follow any regulated diet, no matter how liberal it might be. Some of them made no secret of their refusal to follow directions, but others tried (often successfully) to pass themselves off on their doctor as model patients. About 12 years ago I began to approve of the abandonment of strict dietetic control for those patients who found that they could succeed in maintaining a sugar-free urine only at the cost of hypoglycaemic attacks. Finally, during the last eight years I have never (except in the case of diabetes of very recent onset) advised a diabetic patient to eat a regulated diet, and I have often transferred a conscientious but worried patient from controlled to uncontrolled diet without deterioration in his condition. At the present time none of my diabetic patients are on a controlled diet.

General Principles of Treatment

My experience has been that in all cases of diabetes seen by me the maximum attainable degree of health and vigour could be maintained despite a persistently high blood sugar. The chief aim of diabetic treatment seems therefore to be not a sugar-free urine or a low blood sugar but the maintenance of the patient in health and vigour. This can be accomplished in severe diabetes only by insulin. The object of regulation of meals should be not the maintenance of a carbohydrate balance but the avoidance of hypoglycaemic attacks. Glycosuria implies a regrettable waste of good foodstuffs, but an abundant glycosuria is not incompatible with sustained health and vigour.

Overriding all rules should be the maxim that the right line of treatment for each individual diabetic is that line of treatment on which he is best able to maintain health and vigour over a prolonged period. A lifetime of study may be necessary to make sure that years of apparent good health do not conceal the insidious development of arteriosclerotic or degenerative processes. For that reason I make a plea for the prolonged study of the group of persistently hyperglycaemic (but otherwise healthy) diabetics, for in this study lies the final justification or refutation of the need for strict dietetic control.

Insulin

Insulin in solution is usually given in preference to insulin suspension, the chief reason for the preference being that it demands less complicated instructions as to the prevention of hypoglycaemic attacks. In most cases of severe diabetes in adults two injections daily are found to suffice, but in many children a third (evening) injection is required to prevent a morning ketosis. The total daily dose varies with different cases between 10 and 60 units. If the dose is too low polyuria will probably attract the patient's attention, the urine at some periods of the day will contain ketones, and the weight will fall. If the dose is too high hypoglycaemic attacks will occur in spite of the fact that the patient is eating freely and frequently.

Patients are taught to weigh themselves often and to report at once any substantial change of weight. They are also taught

that an illness of any sort (especially a pyrexial illness or an illness accompanied by vomiting or dysphagia) must be looked on as a serious threat to life entailing probably immediate admission to hospital.

Diet

The first rule taught to a diabetic is that, while no harm follows an occasional omission of the insulin injection, very grave harm will follow if insulin is injected and no accompanying meal taken. This warning makes it clear to him what he should do if compelled by emergencies to miss a meal. The second rule is that a small carbohydrate meal, which need be no more than a hunk of bread, should be taken between each of the three chief meals of the day. The diabetic taking insulin at breakfast and after his midday meal should always have something more to eat in the middle of the morning and in the middle of the afternoon. This rule safeguards him from occasional attacks of hypoglycaemia, especially those which occur from inevitable postponement of one of his chief meals. The third rule is that unwonted bodily exertion should always be followed by ingestion of a little carbohydrate. This precaution is particularly important for those engaged in intermittent heavy manual work or in athletics.

When the patient is first admitted to hospital the state of coma or severe ketosis so often present on admission is corrected, and then he is allowed to eat as he likes. The calorie content of his diet is calculated in order to observe (and if necessary correct) any marked eccentricities of personal taste, and to make sure that it is not inadequate. As a rule two daily injections of insulin of from 10 to 30 units each are given, and this trial dose is increased if there is not a steady gain in weight or if ketosis appears. Usually at some period the patient (unless elderly) is deliberately allowed to experience a mild hypoglycaemic attack.

Urine-testing is never taught, and those patients who insist on testing their urine are cautioned that a sugar-free urine may be a warning of hypoglycaemia.

Most of my patients eat a diet with a high carbohydrate and a minimal protein content. Gluttony is almost unknown. Many are engaged in fairly heavy manual work and have very irregular hours of duty.

Coma

No clinic has been able to produce statistics of recovery so good as those of Joslin, and his doctrine of plenty of insulin and plenty of intravenous saline summarizes, I am convinced, the decisive measures in the treatment of coma or threatened coma. During the first few decisive hours of treatment insulin and saline are administered in amounts dependent on the rate of improvement in the patient's mental state, respiration, and circulatory condition. During these few hours there is little risk of hypoglycaemia. But if the dosage of insulin has been high hypoglycaemia may readily occur at a later stage, for the duration of effect of a large dose of insulin is much longer than that of an ordinary maintenance dose.

I have found that, once the acute stage of coma has been corrected, there is nothing to be feared from the free administration of glucose in amounts considerably above what is required to prevent hypoglycaemia. Coma must often be treated without facilities for blood-sugar estimations, and it seems to me that the permission to administer glucose freely is an essential simplification of treatment; the physician cannot allow himself to be in the position of feeling uncertain whether cerebral disturbances are due to ketosis or to hypoglycaemia. The young doctor must be taught that it is the profound metabolic disturbance in which untreated diabetes culminates that is the cause of coma, and not the accompanying hyperglycaemia; otherwise he may well look on the precautionary administration of glucose to a patient under treatment for coma as a dangerous procedure, or, even worse, may hesitate to give heroic doses of insulin for fear of a hypoglycaemia which he may not succeed in controlling.

Children

The problem of managing the diabetic child is obviously simplified by allowing frequent feeds of normal composition and administering insulin often enough and in large enough doses to prevent ketosis and achieve a gain in weight. But in one important respect the position as regards dieting in children is different from that in adults. Any child, whether diabetic or not, may overeat grossly on occasion, filling his stomach till he vomits. Such "gorges" in the diabetic child are usually the cause of a stormy attack of coma, not, I suspect, provoked by the absorption of too much carbohydrate but by the vomiting. My impression is that the diabetic child to whom the tasty foods of normal children are not forbidden is less likely to indulge in occasional outbreaks of gross gluttony than the controlled diabetic, but if he does indulge he will suffer for it just as certainly.

Acute Diabetes

It is possible that occasionally diabetes is seen at so early a stage that prompt treatment will avert degenerative changes in the pancreas. Though of course it is not easy to prove that the rare cases of complete recovery under treatment might not have recovered spontaneously if untreated, it is obviously wise in all cases of very recent onset to insist on complete rest in bed and very careful control of the blood sugar by means of insulin and a measured diet; only when the passage of time has made it clear that the condition is an established one should the patient be allowed to resume normal activities and an uncontrolled diet.

The Mild Diabetic

By a "mild diabetic" is meant a patient who is able to maintain as high a level of health and vigour without insulin as when taking insulin. Putting aside the regulation of the diet for such conditions as obesity, my experience is that these patients, the elderly, the obese, the arteriosclerotic, and the hypertensive, are none the worse for a high blood sugar. I am very doubtful of the wisdom of restricting the intake of the mild diabetic. In some such patients a sugar-free urine can be achieved only at the cost of under-nutrition. Yet such patients, if allowed a normal diet, may lead an active life without the help of insulin. The borderline patients, who can live without insulin but are better on insulin, sometimes insist on dropping it for a spell. It is my experience that, when they do this and later consent to start injections again, their (small) insulin requirements are not increased by the period of omission.

It is of course important to warn every mild diabetic of the possibility that acute intercurrent illness may bring about a rapid change for the worse and precipitate diabetic coma.

Complications

When the complication is one the danger of which lies in its tendency to provoke coma—e.g., septic infection—the same line of treatment which is successful in the treatment of coma may be applied to its prevention. When the diabetic has to undergo a major surgical operation the physician's attention should be directed not towards keeping the blood sugar low but towards averting ketosis. Tuberculosis is, in my experience, neither more nor less difficult to treat than in the normal subject.

The aetiology of the retinitis, the cataract, and the arterial thickening so common in elderly diabetics is still unknown. There is not, I think, any evidence as to whether such complications are commoner in patients with persistent hyperglycaemia than in the controlled group. But the evolution of these lesions, once they have appeared, does not seem to be affected by the level of the blood sugar. In so-called diabetic gangrene the level of the blood sugar does not appear to have any effect on the progress of the lesion.

Discussion

Against the conclusion which I have drawn from the sustained good health of diabetics whose diet is not controlled—namely, that adequate insulin dosage and the avoidance of hypoglycaemia are the only essentials in the treatment of diabetes—several arguments may be advanced.

1. It may be argued that the health of my patients would have been still better if their blood sugars had been lower. In reply to this it should be noted that the change from a

measured to a free diet has often been made without deterioration in health or vigour.

2. It may be argued that the patients believed to be eating an uncontrolled diet were in fact unconsciously eating a diet which maintained a fairly accurate carbohydrate balance. This is not so.

3. It may be argued that the insulin requirements of my patients are greater than they would be on a measured diet. This may be so, but, as none of my patients need more than 60 units a day and the majority take between 10 and 40, the point is not very important. (This point would be important if it could be shown that cases of established diabetes under control gradually progressed towards complete cure. This question has already been dealt with in the paragraph on acute diabetes.)

4. It may be argued that in 20 years' time the fate of the surviving uncontrolled diabetics may be worse in respect of late complications than that of the controlled group. That is certainly possible. It is no answer for me to claim (as I do) that over 10 years' observation reveals no evidence of premature arteriosclerosis, for it may be that in the development of late diabetic complications there is a clinically silent stage analogous to that in the development of syphilitic aortitis.

If it were not for this objection I would have no hesitation in proclaiming that the maintenance of a carbohydrate balance was an unnecessary adjunct to the treatment of diabetes. But time will settle the question. Twenty years have already elapsed since the isolation of insulin, and soon many young diabetics treated in the first decade after its introduction to therapeutics will be reaching middle age. It will then be possible to judge between the rebellious and the docile, and for that very reason I wish to repeat my plea for a continued careful observation of the former class.

Summary and Conclusions

A plea is made for the study of the group of severe diabetic patients who refuse to follow any regulated diet but will take insulin. Prolonged study of these uncontrolled but otherwise healthy diabetics will show whether their persistent hyperglycaemia favours the onset of arteriosclerotic or degenerative complications. Observations during a period of over ten years yield no evidence that it does so.

Study of this group has convinced me that regulation of the diet with a view to maintaining a normal blood sugar is unnecessary; this opinion is based solely on the sustained good health of patients on uncontrolled diet. The patient must take enough insulin to prevent ketosis, and arrange his meals so as to avoid hypoglycaemia. If he does this his condition is not improved by maintaining a carbohydrate balance. The mild diabetic who does not require insulin does not benefit (unless obese) from strict dietetic control.

In diabetes of very recent origin it should, however, be assumed that accurate dietetic control will favour whatever chance of complete cure may exist.

Normal (uncontrolled) diet in the child is not associated with frequent attacks of coma. Outbreaks of gluttony in the diabetic child are dangerous and lead to coma, not because they produce hyperglycaemia but because they are followed by vomiting.

In the treatment of coma insulin and intravenous saline are the two essentials. If at any stage the onset of hypoglycaemia is feared, then glucose can be administered freely and without blood-sugar control.

Two grants of £1,000 each have been made by King Edward's Hospital Fund for London to assist in the establishment of two group preliminary training schools for nurses—one at Hampstead to serve a North London group of hospitals, and the other at Blackheath to serve a group south of the river. Additional financial help will be given during the next three years should such further help be necessary. The King's Fund is prepared to consider similar schemes by other groups of hospitals in its area and to make grants up to a total of £5,000 for this year, with assurance of continued help for three years in case of need. Further particulars may be obtained from the Nursing Recruitment Centre, 21, Cavendish Square, W.1.

TREATMENT OF ASTHMA BY NICOTINIC ACID

BY

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Maisel and Somkin (1942) have recently published a preliminary report on the treatment of the asthmatic paroxysm with nicotinic acid. Relief of the paroxysm was obtained in 21 out of 30 cases treated with the drug, either by intravenous injection (16 out of 21 patients relieved) or orally (5 out of 9 patients relieved). Eighteen patients who had received intravenous therapy were subsequently given the drug orally, and 11 of these cases were definitely improved. Neuwahl (1942), who had obtained satisfactory results with the drug in angina pectoris, also used it in 7 cases of asthma in order to note its action on smooth muscle. Of his patients 4 were improved and 3 were made worse. It is not known whether the drug acts by relieving bronchospasm or by affecting the pulmonary vessels, or whether it is converted into or liberates another substance which is the physiologically active agent. Neuwahl suggested a pharmacological similarity to histamine; but, as Maisel points out, this does not appear to explain its action in asthma.

For some months before either of these reports came to my notice I had been treating asthma with nicotinic acid, both during the paroxysm and between the attacks. It was hoped that its action in dilating blood vessels might be accompanied by a similar action on the bronchioles.

Treatment of the Paroxysm

Of 19 cases which received treatment during the paroxysm 7 had had slight or no relief from adrenaline or had relapsed. Most of the attacks treated were severe ones. The possibility was considered that improvement following the treatment might be due to psychological influences associated with coming into hospital and receiving injections, and an attempt was made to control this factor by giving an intravenous injection of sterile water, preferably before nicotinic acid, to 12 of the cases. In some instances a control injection was given in a later attack. The effect of the control injection was then compared with that produced by nicotinic acid. Slight transitory improvement was noted in 7 cases after the use of sterile water. Two patients not included in this series had their attacks completely relieved though they did not receive nicotinic acid. In order to be sure of a rapid action, nicotinic acid was as a rule given intravenously in doses of 50 mg. (half those used by Maisel). One ampoule was dissolved in 10 c.cm. of sterile water and the injection was given very slowly. Two cases received intramuscular injections, and in one of these the dose employed was 100 mg. One case received 50 mg. intravenously and 50 mg. intramuscularly. A second intravenous dose was given to many of the patients during a relapse or a succeeding attack, sometimes within a few hours of the first dose. Six cases, 4 of which had already received nicotinic acid intravenously, were given the drug orally in doses of 50 or 100 mg. (1 or 2 tablets) during a paroxysm.

Results.—The effect of nicotinic acid in 16 of these patients was to produce definite improvement in at least one of the attacks for which the drug was given. In 8 cases the relief was practically complete; in the other 8 there was a residual wheeze for many hours. In all cases adventitious sounds persisted in the lungs—a fact noted by Maisel. Improvement usually began to manifest itself within a few minutes of the injection, the patient falling asleep in many instances and awaking the next morning much relieved. The duration of relief varied considerably and did not always appear to be dependent upon further doses, parenteral or oral. The period of relief was sometimes as short as half an hour, but in other cases there was no further attack for some days, even when the attacks had been frequent before admission. On the whole a relief of several hours was obtained. Further injections sometimes repeated the improvement resulting from the first one, but in 5 cases later injections resulted in little or no

relief. This suggests that in some instances the efficacy of nicotinic acid is affected by repeated use, contrary to what is stated by Maisel. It is of course possible that the smaller dosage employed by me might in part explain this difference. Two cases were not improved at all by nicotinic acid. Neuwahl refers to severe exacerbations of asthma which occurred in 3 of his cases. This effect was also seen in 2 cases of this group. One case developed a status asthmaticus 24 hours after an intravenous injection which had given considerable relief and had been followed 20 hours later by an oral dose.

Flushing, tingling, and a feeling of heat appeared within a few minutes of the administration of nicotinic acid, and these symptoms were often pronounced and preceded improvement of the asthmatic attack. Occasionally headache, giddiness, depression, palpitations, and increased tightness of the chest were noted. As Maisel observed, improvement sometimes follows a bout of coughing.

Treatment between the Attacks

In 30 cases, many of which had severe and frequent paroxysms not controlled by ephedrine or stramonium, nicotinic acid treatment was given between the attacks for periods varying from a few weeks to several months. Of these patients 11 had received treatment previously during an acute paroxysm. The drug was given orally generally in doses of 50 mg. two or three times daily. Some cases received 100 mg., particularly on retiring. Children usually received half these doses. It should be noted that Maisel employed oral doses of 200 mg. 3 times daily before meals and once on retiring, but no mention is made over what period such dosage was given.

Results.—The results from this treatment were on the whole favourable. Sixteen cases showed definite improvement as compared with their recent condition before beginning nicotinic acid. This improvement was characterized by a definite reduction in either the frequency or the severity of the attacks and often by both these factors. The period of improvement varied considerably, from a few weeks to several months. Ten other cases showed slight improvement, assessed on the basis of a reduction in the frequency or severity of the attacks or upon the fact that relapse occurred within two weeks of starting treatment. In 4 cases no material change resulted from nicotinic acid therapy. In 9 cases courses of nicotinic acid were alternated with courses of citric acid tablets, the latter acting as a control. The patients were not informed that any change over to different tablets had been made.* In 6 of these cases the effect of nicotinic acid was definitely superior to that produced by the control tablets, the patients generally showing an early relapse when receiving the latter. Two cases, however, were maintained on the control tablets for many months with infrequent attacks. Six cases relapsed rapidly when they were taken off nicotinic acid and received no treatment.

Flushing and the other symptoms produced by nicotinic acid were often present in this group, and two patients abandoned their treatment because of unpleasant effects. It should be noted that several patients in whom the effect of nicotinic acid had worn off responded subsequently to adrenaline and ephedrine, but some were refractory to all treatment.

Conclusions

These results on the whole confirm those of Maisel and Somkin that nicotinic acid appears to be of value in the treatment of the asthmatic paroxysm. The drug when given over long periods also appears to be useful in diminishing the frequency and severity of the attacks. It is necessary to be cautious, however, in assessing the value of any treatment in this condition with such irregular incidence of attacks and susceptibility to psychological influence. Improvement after control injections and tablets illustrates this point. Nevertheless, in the majority of cases in which control therapy was employed, nicotinic acid proved much superior to the control measures in relieving the paroxysms and reducing their frequency. In view of the larger doses given by Maisel and Somkin, it would certainly seem an advantage to increase the dosage above 100 to 200 mg. daily in those cases which do not improve. Yet, although the drug appears to be non-toxic, it can on occasion produce unpleasant symptoms, and the possibility of an exacerbation of the asthma (not noted by Maisel) must be borne in mind. I agree with Maisel and Somkin that at present nicotinic acid should only be regarded as an adjuvant in the treatment of asthma, particularly when adrenaline and ephedrine are proving ineffective or when ephedrine cannot be taken because of toxic symptoms. Further trials with the drug are obviously necessary.

* The absence of flushing, etc., with citric acid did not appear to be regarded by the patients as indicating a change of treatment.

Summary

Nicotinic acid was given to 19 cases of asthma during one or more acute paroxysms in doses of 50 or 100 mg., as a rule intravenously. Definite improvement was obtained in 16 cases. Two cases had marked exacerbations. Control injections of sterile water were usually much less effective.

The drug was given over a long period in 30 cases, 11 of which had already received intravenous therapy. The frequency and severity of the attacks were definitely reduced in 16 of these. The doses used were 50 or 100 mg. 2 or 3 times daily. Half these doses were administered to children. The cases which improved on the drug frequently relapsed when it was discontinued or when they were receiving control tablets of citric acid.

I would like to thank Dr. W. Allen Daley, Medical Officer of Health to the London County Council, for permission to publish this report.

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MENINGITIS DUE TO DIPLOCOCCUS MUCOSUS*

BY

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McFarlan (1941) has recently described the characters of a capsulated Gram-negative diplococcus which had been isolated from the purulent cerebrospinal fluid of a young man. The man had been first seen on account of bed-wetting and slovenliness, and was found to have scanning speech and intention tremor. Pyrexia and signs of meningitis supervened, but he made a rapid recovery after sulphapyridine treatment. The organism was identified as *Diplococcus mucosus*, originally described by von Lingelsheim (1906, 1908) and isolated by him from the naso-pharynx and cerebrospinal fluid. Cowan (1938) had described similar strains isolated from the cerebrospinal fluid of patients after operation for cerebral tumour.

While there is considerable evidence that certain of the nasopharyngeal Neisseria may under favourable conditions give rise to meningitis, the pathogenicity of the capsulated diplococci for man has not been conclusively proved. It therefore seems of interest to report a case in which the causative organism was almost certainly *Diplococcus mucosus*.

Clinical Features

The patient, a man aged 29, with two years' military service, is a driver attached to a field ambulance. On May 24, 1942, he developed a slight sore throat, and the next day a pink macular and papular rash was noticed on the trunk. His throat improved, but he felt vaguely out of sorts during the whole week and the rash persisted. There was no fever during this period.

On May 31 he became suddenly ill with severe frontal headache, pains in the back and limbs, and fever. On admission to hospital a few hours later the temperature was 102° F. and pulse 100. The patient was confused and restless, and complained of intense headache. A profuse pink maculo-papular rash was present on the chest, abdomen, upper part of back, and proximal part of the limbs. No petechiae or purpuric patches were seen. Neck rigidity was present, but Kernig's and Brudzinski's signs were negative. A left divergent strabismus and left-sided ptosis were present, but it was subsequently

* A report to the Medical Research Council.

found that the strabismus had existed since childhood. The pupils were dilated but reacted normally to light. Abdominal and plantar reflexes were normal. There was a leucocytosis of 12,400. Lumbar puncture soon after admission yielded a turbid fluid under increased pressure.

Sulphapyridine was given by the mouth, the initial dose being 2 g. Oral administration was satisfactory, and 9 g. were given in the first 24 hours. The patient rapidly became quieter, and the temperature had fallen to 99.4° F. by the following morning. The general condition was better, the headache less severe, and the mental state normal. The rash had almost completely faded, but petechiae were noted on the anterior aspect of the legs and there was a circular purpuric patch an inch in diameter on the right calf. The ptosis persisted and neck rigidity was still present, but Kernig's sign was negative. Lumbar puncture again yielded a turbid fluid; the pressure was normal. Sulphapyridine was continued in a dosage of 1 g. every 4 hours.

Improvement continued, and during the next few days the rigidity, ptosis, and headache abated. The dosage of sulphapyridine was decreased and the drug was discontinued on June 5, a total of 22 g. having been given. A slight right otitis externa, noticed on June 8, responded quickly to treatment. Convalescence was rapid, and no abnormality was detected on routine examination on June 14.

Laboratory Findings

The cerebrospinal fluid obtained on May 31 was turbid. There were 330 cells per c.mm., mainly polymorphs; protein 240 mg. per 100 c.cm. Fair numbers of Gram-negative diplococci were present, sometimes intracellularly. The opposed sides of the cocci were flattened, but the organisms were on the whole rather larger and more irregular in size than meningococci. Culture of the deposit on blood agar aerobically produced in 24 hours a profuse growth of yellowish mucoid colonies consisting of encapsulated Gram-negative diplococci. The characters of these organisms were as follows:

Morphology.—Gram-negative cocci occurring in pairs with the opposed sides flattened. A capsule was demonstrated by nigrosine and Muir's method.

Growth in Broth.—Diffuse turbidity in 24 hours at 37° C. aerobically.

Agar and Blood Agar, 24 Hours Aerobically.—Abundant growth of low convex, opaque, greyish-yellow round colonies, 2 to 3 mm. in diameter, smooth surface, entire edge, mucoid consistency, undifferentiated. There was no true haemolysis, but the blood underwent a brownish discoloration. Growth was slightly better at 37° C. than at 22° C.

MacConkey's Agar, 24 Hours Aerobically.—Good growth of similar colonies, tending later to develop an effuse periphery and a finely crenated edge.

Gelatin Stab.—Growth along the line of the stab and on the surface. No liquefaction in 3 weeks.

Biochemical Reactions.—Acid, without gas, was produced in glucose in 24 hours, but not in lactose, mannite, sucrose, or dulcite. Litmus milk was unchanged, indole was not produced, and the M.R. and V.P. reactions were both negative.

Pathogenicity.—1 c.cm. of a 24-hour broth culture was injected intraperitoneally into a guinea-pig without effect.

The cerebrospinal fluid obtained on June 1 was still turbid and contained smaller numbers of pus cells, but no organisms were seen in direct films. No growth was obtained on culture. Protein was 80 mg., chlorides 700 mg. per 100 c.cm.

The organism agrees in all respects with the type A form of *Diplococcus mucosus* described by McFarlan, who has discussed the systematic position of these strains and suggests that they are probably variants of the *Neisseria pharyngis* group. Though the evidence points strongly to the present strain being the cause of the meningitis, it would have been more conclusive if the isolation had been made on more than one occasion. Response to sulphapyridine was so prompt, however, that no organisms were found in the second specimen.

We are indebted to the Director-General, Army Medical Service, for permission to publish this report.

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MULTIPLE MUSCLE HERNIAE

BY

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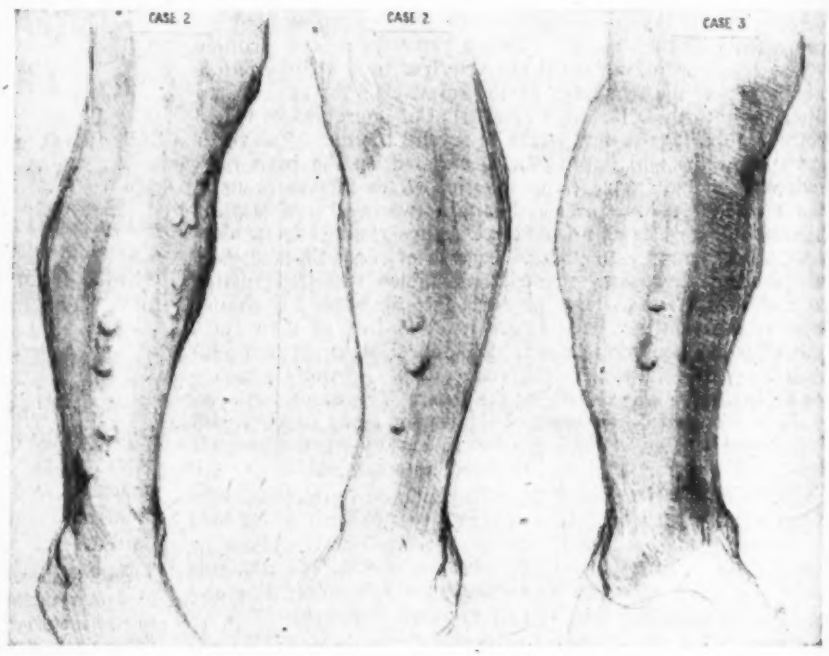
Although from time to time one sees simple muscle herniae, usually of the tibialis anticus, they give rise to little comment, as frequently they cause only slight if any trouble. In most standard textbooks the subject is dealt with very briefly, and few references can be found to it in recent literature. The cases that have been described are mostly those of fairly large single hernia, but bilateral herniae of the tibialis anticus have been recorded.

Ihde (1929), in a comprehensive review of the literature, himself reports 12 new cases, but all those mentioned in the review, including his own, were either single or single bilateral herniae. No case of multiple or multiple bilateral herniae of the lower leg was mentioned. Neither have we been able to find any reference to this condition in later literature. We therefore consider the following three cases of multiple herniae of the tibialis anticus worth recording.

All three patients were soldiers and had been engaged in strenuous training in rough hilly country, and it seems possible that both the nature of the training and that of the country were contributing factors in producing the herniae. This is in accordance with the experience of Hesse (1921), who found muscle herniae rare except in mountainous districts, and of Pichon (Ihde, 1929), who found 39 cases in 217 Alpine soldiers, but only 1 in 1,000 Russian recruits. The herniae we have seen were small—about the size of a pea. All were reducible, and they disappeared when the muscle was contracted, leaving no palpable thickening. The splits in the fascia were distinctly felt. The openings were round and had quite sharp defined edges.



Records of Three Recent Cases

Case 1.—An infantryman was referred to hospital because of aching in both legs, which started during an intensive training course and was attributed to small varicose veins. Previously he had had no trouble, and was not a recent recruit. In addition to the varicose veins, three small reducible muscle herniae about the size of peas were present in the left leg, situated over the belly of the tibialis anticus muscle. The herniae were not tender, and disappeared completely on pressure and when the muscle was contracted.

Case 2 (illustrated).—This patient, an infantryman, was referred to hospital because of aching in both legs and both feet, attributed to varicose veins. He had had no previous trouble, was engaged in a course similar to that of Case 1, and was not a recent recruit. He had no varicose veins, but there were 13 small herniae of the right and 4 of the left leg, all situated over the tibialis anticus muscles. All were reducible, and all disappeared when the tibialis anticus muscles were contracted.

Case 3 (illustrated).—This patient was referred to hospital because of varicose veins. He also was an infantryman and not a recent recruit, and he complained of a "heavy feeling" of the right leg when running. This was first noticed two months previously after a football match, and at that time he observed a small swelling on the front of the leg. Recently he had been taking part in cross-country runs for the first time, and after one run noted the appearance of a second swelling. He had no varicose veins, but there were two small muscle herniae over the tibialis anticus, differing in no way from those in the other two cases.

Discussion

The pathology of muscle hernia is discussed at length by Ihde in his article, and there seems to be no reason why the origin of the small multiple herniae we have found should differ from that of the larger ones which he describes as due to severe strain, with hypertrophy of muscle and weakening and splitting of a previously deficient muscle fascia, as distinct from those which follow direct injury. The only condition with which these small herniae might readily be confused at first sight is varicose veins, but on closer examination the diagnosis is fairly obvious.

One reference only has been found to treatment of a small hernia in this region. Schmier (1937) describes the case of a runner with bilateral herniae over the tibialis anticus muscles, but he does not state the size of the swellings. However, as the man was first seen at a varicose veins clinic they were probably small. Both herniae were treated with repeated injections of sodium morrhuate. This allowed return to strenuous activity without discomfort for a year, when further injections were required to relieve the returning pain; it is doubtful, however, if this method would be suitable for multiple herniae. In the cases we have seen, apart from suggesting modification of duty, no treatment was advised, as the men complained of pain only after strenuous cross-country journeys; otherwise they were fit for all ordinary duties.

It may be that on account of their rarity these multiple small muscle herniae of the lower leg are of no great significance. On the other hand, having seen three cases within a few weeks in soldiers undergoing intensive training we considered them of sufficient interest to warrant a brief report.

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Medical Memoranda

Mantoux Test versus Patch Test

Several papers have been published in recent years on the detection of tuberculous allergy by the patch test. In Monrad's (1936) series of positive Mantoux reactors 5% were negative to the patch test; in Wolff and Hurwitz's (1937) series 1.8%; in Vollmer and Goldberger's (1937) less than 1%; Hart's (1938) first series had 3%, and his second (1941) less than 2%, while Court's (1939) series had 2%. Camille Kereszturi (1941), however, showed that 15% of his positive Mantoux reactors were negative to the patch test. It is advisable, therefore, to check up on these findings from time to time, and for this reason the results of an investigation seem worth recording.

Investigation of 116 Cases

All the children admitted to the medical wards of a base hospital over a certain period were tested by the intradermal (Mantoux) and the percutaneous (patch) methods. The age varied from 6 months to 12 years. In the intradermal test 0.1 c.cm. of 1 : 1000 freshly prepared old tuberculin was injected into the skin of the forearm: this was sufficient to raise a wheal 8 mm. in diameter. The test was read 24 and 48 hours after injection, and was not considered positive if the resulting wheal or flare was less than 10 mm. in diameter. In carrying out the percutaneous test a tuberculin patch was applied to the chest wall between the scapulae and lateral to the spine after cleansing the skin with ether and allowing it to dry. The patch was removed after 48 hours, and the reaction was read 24 and 48 hours after the removal. Papules or a red flare, or any intermediate stage, were regarded as a positive reaction. The patches used were those of Messrs. Evans Sons Lescher and Webb. The following were the results:

Total number tested	116
Number negative to both tests	74
,, positive ,, ,, ,,	34
,, ,, to intradermal and negative to patch test	8
,, negative ,, ,, ,, positive ,, ,,	0

There was one case of plaster dermatitis. In two cases the patch tests were negative after 24 but positive after 48 hours. These were considered positive reactions. Of the Mantoux tests 10 were apparently positive after 24 hours, but had faded to negative after 48 hours. These were recorded as negative reactions.

Summary and Conclusions

The intradermal Mantoux test and the tuberculin patch test were performed on 116 children.

The Mantoux test was positive in 8 cases in which the patch test was negative.

Both tests should be read 48 hours after being applied, to avoid false positives thought to be due to the erythema around the needle puncture. It is important to use a preparation of old tuberculin not more than three weeks old.

It seems likely that different observers employing patches of different make may get variable results. The intradermal method is less liable to diversity and is more reliable.

I am grateful to Dr. Wyllie for his interest in this investigation, and for permission to publish the series; also to the nursing staff for their helpful co-operation.

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Convulsive Treatment of Climacteric Depression

Treatment of cases by electrically induced convulsions was begun in this hospital in March, 1942, and for the nine months ending Dec., 1942, we had the following results in female patients:

	Acute	Chronic	Total
No. of patients treated	43	17	60
Total No. of convulsions	945	349	1,294
No. of patients discharged	*30 (69·9%)	0	30

* Of this total 26 (60·6%) were discharged recovered and 4 (9·3%) were discharged relieved.

The average stay in hospital of all discharged cases was 5½ months—in recovered cases 5½ months, and in cases of involutional melancholia 4 months. The classification of these patients, the number treated, and the percentage rate of discharge in each class were:

Diagnosis	Number	Percentage Discharge
Involutional melancholia	25	68
Schizophrenia	19	31·5
Mania	3	100
Psychoneurosis	3	100
Other psychoses, including mixed types, puerperal confusion, and paraphrenia	10	30

The results in schizophrenia are poor. The 31.5% discharge rate noted in this group occurred in cases of very recent onset, and their clinical histories suggested the likelihood of remission with or without special treatment. The course of the illness noted under mania, psychoneurosis, and "other psychoses" seemed also to be unaffected by electrically induced convulsions.

As with other workers, our experience suggested that the best results are to be obtained in melancholia, especially of the involutional type. According to Jessner and Ryan (1942), "rather striking results have been reported in involutional

melancholia." This is not surprising, since the case records indicate that climacteric depressives have the most favourable prognosis of our female admissions. It seemed important, therefore, to consider whether treatment by electrically induced convulsions shortened the length of stay in hospital.

The length of illness before the use of this method of treatment was approximately six months. In the group noted above the patients were discharged recovered after an average stay of four months. It would thus seem a reasonable inference that treatment by electrically induced convulsions accelerates discharge of mental hospital patients who are admitted suffering from recoverable depression. Whether this recovery is associated with a greater or less degree of permanence it is too early yet to determine. So far, however, the reduction of time lost in illness from six months to four makes a strong case for the value of this form of treatment for this particular type of case. It has, too, the advantage of comparative safety. In 1,300 convulsions there were only two minor accidents: one a dislocation of the jaw, and the other a subluxation of the shoulder-joint.

I wish to thank Dr. George Somerville, medical superintendent, West Ham Mental Hospital, for permission to publish these results.

West Ham Mental Hospital. ELIZABETH P. DODDS, M.B., Ch.B.

REFERENCE

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Treatment of Intestinal Carriers of the Enteric Group of Organisms

The following note on the results of the use of sodium iodophthalein in enteric infections may be of interest.

CASE HISTORY

A girl aged 16 was admitted to the local fever hospital on Sept. 24, 1941, after an illness of ten days. She complained of abdominal pain with constipation. The temperature was 98.8° and the pulse 90. Some ten to fifteen typical "rose spots" were present over the chest and abdomen, and there were one or two on the back. The following day blood and faecal specimens were sent for bacteriological examination. The Widal reaction was positive to *Bact. paratyphosum* B infection, while the faecal specimen showed the presence of the same organism. Progress and convalescence were uneventful. Her stools, however, examined at weekly intervals, persisted in giving growths of the organism for 22 weeks despite the use of such drugs as hexamine, salicylates, bismuth c salol, and vegetable carbon.

Reading of Saphir and Howell's (1940) successful treatment of a carrier of *Bact. paratyphosum* A with sodium iodophthalein (opacol), it was decided to try the effect of the drug on this carrier of *Bact. paratyphosum* B. The technique and dosage were identical with those used in *x*-ray examination of the gall-bladder. Two doses were administered, with an interval of a fortnight. The faeces remained positive after the first dose, but after the second dose three specimens taken at five-day intervals were returned negative.

The patient was discharged on March 13, 1942, after spending 24 weeks and 3 days in hospital—i.e., 26 weeks after the onset of the illness.

Since treating the above case I have had an opportunity of watching the effect of sodium iodophthalein in seven cases of *Bact. typhosum* infection. The essential points may be tabulated thus:

Case	Age (years)	Number of Weeks of Infection before Administration of Opacol	Number of Doses Required
1	38	10	2
2	11	14	1
3	13	15	1
4	17	10	2
5	8	10	3
6	23	7	2
7	54	5	2

All the patients were discharged after repeated negative stools.

COMMENT

The above is a method of treating medically intestinal carriers of the enteric group of organisms and should be tried before submitting the patient to any operative procedure.

Two doses of sodium iodophthalein are usually required.

The last two patients were members of the staff and were given the drug early in order that they might return to duty, thus saving man-power.

My thanks are due to Dr. S. K. Young, M.O.H., for permission to publish these cases.

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Reviews

SHOCK

Shock. Its Dynamics, Occurrence and Management. By Virgil H. Moon, M.Sc., M.D. (Pp. 324; illustrated. 21s.) London: Henry Kimpton.

Why the first use of the word "shock" in medicine should be "credited" to this person or that is a mystery: many workers in this field to-day feel that it would be more fittingly debited. Seldom has so much confusion been due to so small a word, and monosyllabic at that: ignorance is usually cloaked in polysyllabic Latin derivations. The ancients from their fleeting glimpses of the unicorn described it with the legs of a buck, the body and head of a horse, and the tail of a lion; but we are still uncertain whether they described one animal or three. Dr. Virgil Moon admits the confusion between "shock" and the effects of haemorrhage, but considers shock to be an entity. In this new book (which supersedes his previous monograph, *Shock and Related Capillary Phenomena*, published in 1938) he believes still that this fabulous concept may be caught in the net of yet another definition. An essential property for catching the unicorn was a virgin, before whom it would lie down and allow itself to be apprehended. The ancients ascribed their notable want of success to the difficulty of finding a virgin. Our own failure with the shock problem, though perhaps partly due to the entertainment of preconceived ideas, can be more properly ascribed to too little of the right kind of experience. Virgil Moon, however, has no lack of experience in this field, and in the space of 324 pages, with the help of 437 references, he tries to tame the animal.

The author discusses at length the role of anoxia, toxaemia, local loss of fluid, nervous stimulation, anaesthesia, and adrenal dysfunction in the genesis of shock, and points out that confusion arises on four main points: lack of knowledge of capillary physiology, failure to pursue pathological inquiries, unrecognized sources of error, and a belief in the identity of shock with the effects of haemorrhage. We would point out a fifth: the application of results obtained by animal experiment directly to man. For instance, he states (p. 262) that "haemodilution occurs *immediately* after haemorrhages," having previously cited two references to animal experiments. If this were so in man, it might be useful in differentiating haemorrhage from some types of "shock," but recent work has shown that it may not occur for several hours. A great deal of stress is laid on capillary pathology—14 out of 19 photographs depict the lung. There is at the moment an unparalleled barrage of investigation directed on the "shock" section of the research front, and many of the views expressed herein will doubtless have to be modified as a result. This in no way, however, detracts from the value of this monograph, which sets out clearly the present point of view of an experimental pathologist, and poses numerous points for energetic argument and examination, together with many useful references.

OPHTHALMIC TREATMENT

The Ophthalmic Prescriber's Codex. By Francis E. Preston, D.O.M.S. (Pp. 176. 10s. 6d.) London: H. K. Lewis and Co. 1943.

In the *Ophthalmic Prescriber's Codex* Dr. F. E. Preston brings together an imposing mass of prescriptions, a guide to treatment, and much pharmacological knowledge. The section dealing with prescriptions covers about one-third of the book and is a full ophthalmic pharmacopoeia interspersed with concise and useful information on dispensing. It is conveniently arranged, and its value is enhanced by the fact that it gives both metric and imperial weights and measures. As this section is probably the fullest ophthalmic pharmacopoeia available, its usefulness can hardly be questioned. Nor can there be any cavil at the concluding 50 pages of the book, which give in readily accessible form such despised but essential things as lists of abbreviations in common use, normal values of various body fluids, posological tables, lists of incompatible drugs, weights and measures equivalents with conversion tables, and much other information besides. There are specially valuable sections on buffered ophthalmic solutions and on the sulphonamides.

It is the middle section, dealing with treatment, that is open to criticism. Those who like tabulated lists of therapeutic

measures will find this section exhaustive enough, but the reviewer would have liked more recognition of masterly inactivity and plain confession of therapeutic impotence. It is, however, only right to stress that the treatments suggested show a wide acquaintance with modern methods, and Dr. Preston no doubt meant this section to be a reminder rather than oecumenical.

NURSING IN THE TROPICS

Tropical Nursing. A Handbook for Nurses and Others going Abroad. By A. L. Gregg, M.D., M.Ch., B.A.O. Second edition. (Pp. 185. 6s.) London: Cassell and Co.

This handbook describes the symptoms, signs, and treatment of the commoner and more important tropical diseases from the standpoint of their practical nursing, the diseases being arranged in alphabetical order so that reference is easy. A section also deals with the technique of the simpler diagnostic and therapeutic procedures that a nurse in the Tropics may be called upon to carry out, for in the Tropics a nurse may have to do many things that at home would normally be performed by one of the junior medical officers. There is also a chapter on personal hygiene, which gives many useful hints to the nurse who may be about to take up her first appointment abroad.

The book is of convenient size, well printed, and free from serious errors. It will be of considerable value to those who—for example, now in military service—may be meeting tropical diseases for the first time, and to those whose work takes them to remote areas abroad where they will have to rely largely on their own knowledge, judgment, and initiative. It is moderately priced and should continue to enjoy its already well-deserved popularity among nurses dealing with tropical diseases.

Notes on Books

LANGDON-BROWN and HILTON'S book *Physiological Principles in Treatment* is now 34 years old, and reading it one has much the same impression as in reading *Hamlet*, that one has heard it somewhere else before. But this is really the highest praise one can give, for it shows that, as a result of this book and others of the same kind, the profession is now more physiologically minded than when Dr. Langdon Brown (as he then was) first wrote it. Indeed the very success of such books may diminish the demand for them. To-day the need is for a greater application of psychological ideas in medical treatment and for more applied physiology in the factory, the armed Forces, and in preventive medicine rather than at the bedside. There are one or two changes we should like to see in future editions. The newer alkalis, magnesium trisilicate, aluminium hydroxide, and aluminium phosphate, receive less attention than they deserve, and the biochemical basis of action of the sulphonamide drugs might have been more fully discussed. It is also doubtful whether wartime decreases in diabetes mellitus are due to restriction of carbohydrate, for there is a good deal to suggest that the incidence of diabetes is more closely correlated with the consumption of fat than of carbohydrate. These are minor criticisms, however, and the book is aimed at a worthy target. It does not make the mistake of Macaulay or the Americans of over-estimating the time and capabilities of the average student, and it tells him just as much as he ought to know if he is to preserve the physiological outlook through his first overwhelming clinical years. The publishers are Baillière, Tindall and Cox, and the price is 12s. 6d.

The appearance of the fifteenth edition of *Stedman's Practical Medical Dictionary* (London: Baillière, Tindall and Cox; 42s.) barely three years after its predecessor is clear evidence of the demand for this well-known reference book. In its preparation the editor, Dr. S. T. Garber, must have had a more than usually arduous task. Not only has the dictionary been brought up to date, but it has been entirely reset in a new type, chosen, after much deliberation and exhaustive tests, for its legibility and clearness—and that it possesses these qualities we certainly agree. The section on that ever-expanding subject the vitamins has been revised, and many new biochemical and chemotherapeutic substances are described. The entire terminology of the bacteria, too, has been revised to agree with the type-species concept incorporated in the International Rules of Botanical Nomenclature, and cross-references are made from the older to the newer titles. Fresh illustrations have been included and some old ones replaced. The introductory article on medical etymology will continue to appeal to the more literary-minded; and another useful section that has been retained is the nomenclature in Latin and English adopted by the Anatomical Society of Great Britain and Ireland, with B.N.A. equivalents. Those who do not allow themselves to be unduly disturbed by American spelling will welcome the latest edition of *Stedman's*.

Preparations and Appliances

SOME USEFUL OPHTHALMIC SULPHONAMIDE PREPARATIONS

Dr. I. LLOYD JOHNSTONE (Worcester) writes:

Considerable attention has been given to the subject of local application of sulphonamides in ophthalmology. The early idea that this class of therapeutic agent was without effect when topically administered is no longer tenable. Without going into a review of the literature reference may be made to the work of Robson and Scott (1942), using sulphacetamide on experimentally produced corneal ulcers, and to an earlier clinical study of hypopyon ulcer effectively treated with sulphapyridine or sulphanilamide powder, or with soluseptasine as drops or by subconjunctival injection (Johnstone, 1941).

A need has been felt for a vehicle for these drugs which would keep them in contact with the diseased tissues in sufficient concentration and yet not insulate them. Such vehicles have been found, but, unfortunately, are in short supply. Messrs. May and Baker have made a sulphathiazole ointment (10%) in a soft wax base, and Messrs. Boots (Nottingham) an ointment of sulphacetamide (10%) in a water-miscible base of the appearance and texture of a cold cream. Both of these products are proving useful in various types of blepharitis, from acute abscess of the lid to the extremely chronic blepharitis with slight redness, itching, and scaliness of the lid margins. A case of dermatitis of the face from constant bathing with tears in a child with phlyctenular keratoconjunctivitis cleared up quickly with the sulphathiazole (thiazamide) ointment after being resistant to other forms of treatment.

Burns of the lids can be treated with the sulphacetamide cream, while burns involving the globe need an oily emulsion of the same drug. Unemul (Universal Emulsifiers Ltd.) has been used as an emulsifier, but there is difficulty in getting a stable emulsion unless a homogenizer is employed. A useful prescription is: sulphacetamide solution (from a 30% ampoule) 10%, water 25%, glycerin 30%, unemul 5%, cod-liver oil to 100%. Atropine sulphate (1%) must be added when the cornea is involved.

The dramatic effect of sulphapyridine in ophthalmia neonatorum will bear emphasis here. One-eighth of a tablet given with or after each feed will clear the condition in 48 hours. The treatment should be continued for a further 24 hours after apparent clinical cure. If this treatment is started early there should be no need to send a case to hospital. Local treatment of the eyes is reduced to a minimum. While discharge lasts it should be gently bathed away with cotton-wool pledgets soaked in warm saline. This should be repeated just often enough to prevent the lids from sticking. A drop of liquid paraffin or of 5% argyrol may be instilled each time, but so effective is the sulphapyridine that the drops can generally be omitted except as a memorial tribute to the past.

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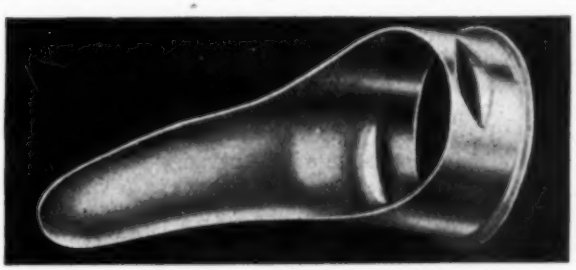
MOUTH GAG FOR LARYNGOSCOPY

Surgeon Commander H. PARRY-PRICE (Banbury, Oxon) writes:

I enclose an illustration of a mouth gag that has been made at my suggestion by Messrs. A. Charles King as an aid to direct laryngoscopy. Some patients have awkward gaps in the upper front teeth, and some difficulty may be experienced when



passing a laryngoscope for direct-vision intubation. There is a gutter into which the teeth fit, and the spatula-like end controls the tongue. I find that with a preliminary spray of 2% percaine, followed by pentothal intravenously, it is perfectly easy to insert this prop and pass the tracheal tube.

BRITISH MEDICAL JOURNAL

LONDON

SATURDAY MAY 15 1943

NERVE REGENERATION

In addition to its intrinsic interest, the subject of nerve regeneration becomes increasingly important in time of war, and it is good to know that British workers are to the fore in attempts to solve the numerous theoretical and practical problems that arise in connexion with it. There is an extensive literature on the subject, as many as 928 papers being collected by Rossi and Gestaldi[1] up to 1934. Most of these lay emphasis on regeneration from the point of view of histology or function, but few studies have combined the two. In a recent review Young[2] has pointed out that the functional recovery of a regenerated nerve becomes complete only when the fibres are of a certain diameter and state of medullation, and can carry impulses of appropriate frequency and velocity. On this premise Holmes and Young[3] put forward six factors which may affect the success of secondary nerve sutures: (1) The first is the power of the central stump to send out new fibres. They find that this power is not affected when the axons are cut twice, either within a few days or nearly a year after the original injury. (2) The second is the power of the stumps to unite. This depends upon the outgrowth of Schwann cells from the peripheral stump. This outgrowth is at a maximum about two to three weeks after section of the nerve. There is evidence that union made after this interval is somewhat more successful than after primary suture. From about 100 days onwards the power of union declines, and long delays of union are less likely to succeed than primary suture. (3) The total diameter of the peripheral stump shrinks in the later stages of degeneration, sometimes by as much as a half. This fact makes a good suture difficult to effect and militates against the successful re-entry of fibres into the peripheral stump. (4) The shrinkage of the lumen of the Schwann tubes results in fewer fibres entering each one, and this in turn diminishes the chance of successful re-connexion. (5) After delayed suture, medullation is delayed in the peripheral stump and the fibres remain of small diameter for much longer than after primary suture. (6) The factor of re-innervation of end-organs is of great importance. Holmes and Young conclude that the process of the union of stumps and the regeneration of a new functionally efficient stretch of nerve are less satisfactory after the longer period of degeneration. Postponement of suture for one or two months does not prejudice the chances of recovery and may even improve them. Longer delays, especially those greater than six months, bring about conditions which are likely at least to retard recovery and may permanently prevent it.

Weddell[4] has correlated both the macroscopical and microscopical appearance of regenerating nerve fibres with the diminution of the area of sensory loss following interruption of nerve in the rabbit's ear. He finds that nerve fibres first advance along the original main nerve fasciculi. The first fasciculi to be re-innervated are those lying in closest proximity to normal nerve trunks. He has also found that cutaneous nerve bundles close to the larger blood vessels are more rapidly re-innervated than those further away. This, it should be emphasized, is distinct from the extension of adjacent normal fibres which also occurs towards the area of sensory loss. When the nerve trunks have become re-innervated the fibres pursue a tortuous course through the cutaneous plexus towards the skin surface. The finest visible endings of the nerve fibres are associated with Schwann cell pathways, which retain the pattern of the cutaneous plexus in the normal ear. On no occasion in the rabbit's ear has the tip of a regenerating nerve fibre in the cutaneous nerve plexus been seen pursuing a course independently of a Schwann band.

In a recent publication Weddell[5] observed that cutaneous sensory spots are each innervated by multiple nerve fibres. He suggests, therefore, that during the course of regeneration these will arrive at each separate spot at different times because they approach it from different directions, and the ultimate courses followed by the individual fibres will necessarily be of different lengths. Thus there will be a phase during regeneration in which each sensory spot is innervated by a single fibre instead of by multiple fibres. Such a stage in regeneration has now been found to occur, and hence the physiological findings of Trotter and Davies[6] are seen to have an anatomical basis—that is, if the process of regeneration of cutaneous nerve fibres in the ear of the rabbit can be assumed to be similar to that in man. The more rapid regeneration of nerve fibres along blood vessels is also in accord with the observations of Trotter and Davies, who observed that vasomotor control returned more rapidly than skin sensation. These phenomena evidently play some part in the process of diminution of areas of sensory loss which are observed clinically to take place from the periphery towards the centre of a degenerated area rather than from the centre outwards. In contrast with this mode of innervation the return of skin sensibility in regions not surrounded by areas innervated with normal nerves, such as the digits, is by an advancing wave of sensory recovery along the line of the cutaneous nerves supplying the skin. A similar phenomenon occurs in the rabbit's ear after complete denervation. The more rapid regeneration of cutaneous nerve fibres in the neighbourhood of blood vessels may serve to explain the bridges of cutaneous sensibility which are commonly found, in man, to cross an area of sensory loss during the course of regeneration from the proximal stump of the divided nerve.

The process of nerve regeneration throughout an area of skin involves first the rapid regeneration of nerve fibres along the main subcutaneous nerve trunks, and it is only at a later date that fibres extend from these trunks towards the skin by way of cutaneous nerve plexuses. In other words, regenerating nerve fibres ramify in the subcutaneous tissues some time before they give extensions to the skin itself. This is important from the point of view of nociceptive sensory tests which are used to determine the progress of sensory recovery. For instance, it has been found in clinical cases that " deep pricks " (2 to 3 mm. deep) will arouse pain in an area which is still anaesthetic to lighter pricks (1/2 to 1 mm. deep). This furnishes an explanation for the statement that one of the earliest indications of recovery of skin sensibility is pain caused by pinching the skin, for this manipulation necessarily includes the subcutaneous tissues. The importance of using standard instruments, which will give approximately constant stimuli from day to day in the same patient, cannot be overemphasized if a true picture of the course of sensory recovery (nociceptive) is to be obtained.

[1] *Rev. Path. Nerv.*, 1943, **43**, 1.
[2] *Physiol. Rev.*, 1942, **22**, 318.
[3] *J. Anat.*, Lond., 1942, **77**, 63.
[4] Ibid., p. 49.
[5] *Proc. roy. Soc. Med.*, 1941, **34**, 776.
[6] *J. Physiol.*, 1909, **38**, 134.

The work reviewed above has demonstrated the process of peripheral nerve regeneration in detail and has accounted in some measure for the clinical observations made on functional recovery. After complete division of a nerve early suture is important. In cases of incomplete division or neurapraxia (Seddon[7]) a most careful assessment of the damage done is essential before operative intervention is decided upon. To this end Guttman[8] has shown the value of the thermoregulatory sweating test, which gives a clear indication of the amount of autonomic damage in the cutaneous nerve. More recently Weddell, Feinstein, and Pattle[9] have shown that an accurate assessment of the injury to a peripheral nerve supplying a voluntary muscle can be made by electromyography. It is to be hoped that by combining these two procedures with a clinical examination the treatment and prognosis of peripheral nerve injuries will advance considerably.

To complete the picture of work in progress on nerve regeneration we will refer briefly to the less familiar problem of fibres in the central nervous system—a matter recently investigated by Le Gros Clark.[10] In 1910 Tello[11] implanted fragments of peripheral nerves in the brain. He claimed that regenerating fibres were seen in the graft, and in some sections could be seen penetrating the perineurium from the tissues of the host brain. Tello concluded that the Schwann cells were a source of a specific neurotropic substance which attracted fibres from cerebral neurones to grow towards them. Le Gros Clark, on the other hand, found a few regenerating fibres within his implanted graft in only 3 out of 13 experimental animals. In two cases regenerating nerve fibres were traced to a fasciculus accompanying newly formed blood vessels which approached the graft from the tissues of the host brain. In one case regenerating fibres within the grafted nerve fragment appeared to have entered the latter from the tissues at the surface of the brain. In no instance could regenerating fibres be traced into continuity with the fibres of the host brain, though the possibility of such a derivation could not be excluded. In some experiments many fibres of the surrounding white matter were disposed in fasciculi orientated towards the graft. This appearance was due partly to traction in the host tissue by the graft and partly to the dislocation of pre-existing fibres by a cellular infiltration. In two experiments spinal ganglia grafted into the brain showed great regenerative activity. Many fibres were found to penetrate into a zone of cellular infiltration separating the graft from the tissues of the host brain. However, no regenerating fibres from the host brain were found to extend into this zone towards the graft. It is concluded from these careful and detailed observations that the intrinsic fibres of the host brain have little if any regenerative capacity compared with peripheral nerves.

Sugar and Gerard[12] reported evidence of regeneration after section of the spinal cord in foetal and newborn rats. However, it is well recognized that the central nervous system of the newborn rat is very immature, and the post-operative growth of nerve fibres may well have been due to the developmental outgrowth of axons which would be proceeding normally at this time. These authors also cut through the spinal cord in young rats aged 3 to 5 weeks and in some cases reported regeneration across the scar. But, as is pointed out by Le Gros Clark, the anatomical evidence for this is not easy to assess; the consistency of the white matter of the central nervous system makes it difficult to produce clean lesions experimentally, and the fibres which appear to cross the scar at the site of lesion may be the frayed ends of the pre-existing fibre bundles pulled out of their normal position and left overlying the line of the knife cut. In addition to this the central nervous system of young rats even a few weeks old is relatively immature. Mitoses in the spinal cord reach their height in white rats about the 7th day after birth. It seems probable, therefore, that axons within the central nervous system do not regenerate once they are mature. It is also clear that the explanation for this is not to be sought in the absence of Schwann cells in the central nervous system but in other factors. Tower[13] has lately shown that the anterior horn cells in the spinal cord, when avulsed, can send out new axons along paths devoid of Schwann cells. There seems to be some factor related to all peripheral nerves which is absent in the intrinsic neurons of the central nervous system.

7 *British Medical Journal*, 1942, **2**, 237.
8 *J. Neurol. Psychiat.*, 1940, **3**, 197.
9 *Lancet*, 1943, **1**, 236.
10 *J. Anat.*, Lond., 1942, **77**, 20.
11 *Trab. Lab. Invest. biol. Univ.*, Madrid ,1911 **9**, 123.
12 *J. Neurophysiol.*, 1940, **3**, 1.

RESPONSIBILITY OF A PSYCHOPATH

The shortcomings of the "rules in M'Naghten's case" were again demonstrated in a recent trial for murder at the Old Bailey (Rex *v.* Lees-Smith). The defence alleged that the accused's act had been due to mental deficiency. Lengthy medical evidence was given concerning a number of laboratory tests which were applied to the prisoner. The witnesses found, in short, that he suffered from a congenital constitutional defect of development, manifesting itself as emotional immaturity, poor physique, and minor abnormalities of the central nervous system. He did not necessarily suffer from epilepsy, and in their opinion he suffered rather from psychopathic personality. They gave evidence that when his blood sugar was low and he overbreathed a change occurred in his brain whereby his judgment was impaired and his full perception of events was distorted. Before committing the crime of matricide he had drunk four pints of beer. A similar quantity given in the laboratory had the effect of lowering his blood sugar and aggravating his abnormality. The medical witnesses concluded, in terms of the M'Naghten rules, that when he killed his mother he knew what he was doing and that it was wrong, but his brain was functioning abnormally so that he was unable fully to appreciate the nature of his act.

The learned judge explained clearly to the jury the meaning of the M'Naghten rules, but in spite of his direction they found the prisoner guilty but insane. Everyone concerned with the case realized that the prisoner would almost certainly have been reprieved on the advice of the Home Secretary. The defect of the M'Naghten rules, which are now just 100 years old, is that they do not provide for the criminal who knows what he is doing and knows that it is illegal but, owing to a diseased or defective mind, cannot restrain himself from doing it. Many murderers have in fact been hanged in spite of strong evidence that their mental state had deprived them of self-restraint, and perhaps as many have been found guilty but insane in face of the M'Naghten rules. These rules were laid down by the common-law judges in 1843, and knowledge of human psychology has much advanced in the last hundred years. In 1924 Lord Darling introduced into the House of Lords a Bill to provide for the defence of "irresistible impulse," but it was opposed by several Law Lords and its mover withdrew it. The general feeling among lawyers seems to be that many criminals who richly deserve death would be able to persuade a jury that they had acted under an irresistible impulse. Persons who feel in this way perhaps underrate the capacity of juries to assess the medical evidence wisely and to reject a plea of this kind if the accused does not clearly show that his mental abnormality

13 *Arch. Neurol. Psychiat.*, Chicago, 1943, **49**, 1.

had robbed him of responsibility. They may also forget that the consequence of such a finding would not be acquittal but detention as a criminal lunatic. In the 1924 debate Lord Haldane said he had never heard of the M'Naghten rules embarrassing any judge when justice required an acquittal, and that there need be no fear that anybody who had acted under an impulse he could not resist was likely to be hanged. This *Journal*, commenting on the matter at the time,[1] said there were surely objections to a procedure by which prisoners are sentenced to death when, as Lord Sumner observed, the exercise of the prerogative of mercy is as certain as that the sun will rise the next day. The same comment is equally applicable to the present case. The law contains too many archaisms of this kind, and in too many important respects has fallen out of touch with reality. Doubtless this is an important factor in the ominous shift of power from the courts to the administrative departments which has marked the last two decades.

THE CONTAMINATED INJECTION

In 1928 a doctor in Bundaberg, Australia, used diphtheria prophylactic from a rubber-capped bottle of toxin-antitoxin which contained no antiseptic. Each time he wanted some more prophylactic he plunged the syringe needle through the rubber cap and took the required amount from what was left in the bottle. The material thus became contaminated with successive use, and of 21 children who were given injections of prophylactic on one day, 12 died of staphylococcal toxaemia caused by staphylococci which had been accidentally introduced into the bottle and had grown there. Bottles containing remnants of the same batch of prophylactic were then obtained from 33 doctors, and the fluid in 10 of these bottles was found to have become contaminated during use. Inadequate cleansing of the cap, non-sterile needles, or germ-laden air were suggested as possible causes. Every doctor in his daily work uses bottles containing several doses of prophylactic, or local anaesthetic, etc. To withdraw fluid through a rubber cap he must relieve the "vacuum" caused by the previous withdrawals; he therefore injects air with his syringe. The Australian Royal Commission, in its report on the fatalities at Bundaberg in 1928, described some experiments with these multiple-dose bottles. Sterile broth was put into sterile rubber-capped bottles, and a quantity of atmospheric air was forced through the cap with a sterile syringe. When this was done in a quiet room there was no contamination of the broth in the bottles, but when it was done in a dusty room with a ventilating fan running, the broth in 6 out of 11 bottles was found to contain bacteria. Air from a dusty room was next blown into bottles containing broth and 0.5% of phenol, but none of the broth showed any bacterial growth. It seems reasonably safe, therefore, to blow ordinary air into material which contains antiseptic in proper amount. Almost all medicaments in rubber-capped bottles have some antiseptic, but this is not enough to kill any spores that may be introduced through faulty technique. To make the procedure as safe as possible—alike for fluids with little antiseptic or none, and in unusually dust-laden air—some simple method might be devised for filling the syringe with sterile air. The sterile syringe needle might be buried in a tightly rolled sterile plug, or pushed through a rubber diaphragm of a sterile glass tube with a sterile stopper at the other end. This may seem a cumbersome procedure, but may be well worth remembering in special circumstances: the Australian doctors in their ordinary daily work contaminated 30% of the bottles of prophylactic.

[1] *British Medical Journal*, 1924, **1**, 922.

VITAMIN C AND CARBOHYDRATE METABOLISM

The relation of vitamin C to carbohydrate metabolism has often been investigated in the past few years. The reports so far are rather contradictory, though the majority of them suggest some sort of a relation. The method of investigation usually employed was a measurement of either the fasting blood-sugar level or the sugar-tolerance curve after administration of the vitamin. Most workers reported a fall of blood sugar; some failed to notice any change, while a few even observed a rise. A change of blood-sugar level resulting from the administration of the vitamin can hardly be taken as conclusive evidence of the latter's relation to carbohydrate metabolism, for the change might be the result of a lowering of renal threshold, or be due to the influence of some other mechanism following administration of the vitamin.

Recently from the Bispeljerg Hospital, Copenhagen, have come two more reports indicating a lowering of blood sugar after administration of vitamin C. The first work[1] involved a measurement, after intravenous injections of vitamin C, of the change in fasting blood sugar in hospital patients with normal blood-sugar levels. A control was obtained by giving the same patients injections of plain water. Vitamin C caused a fall of blood sugar in the majority of the cases examined. The magnitude of the lowering, however, bore no relation to the initial vitamin C content of the blood, nor did it correspond with the variation in the concentration of the vitamin in the blood after injection. As pointed out above, these observations by themselves are insufficient proof that the vitamin is directly related to carbohydrate metabolism. The second paper[2] refers to glucose-tolerance tests on patients at varying levels of vitamin C nutrition. Some subjects whose blood vitamin C was very low showed a diabetic type of glucose-tolerance curve with an abnormally high maximum, which was amenable to treatment with the vitamin. These subjects were distinguishable from true diabetics, whose sugar-tolerance curves were not much altered by treatment with the vitamin, though there was sometimes a slight lowering of the maxima. Hyperglycaemia not uncommonly occurs in infectious diseases, and the suggestion is thrown out that it may be caused by the deficiency of vitamin C which often accompanies infection. These observations still need confirmation in view of the small number of cases studied, and their interpretation should be viewed with reserve. Certainly it would be unwarranted to assume that ascorbic-acid deficiency is the sole cause of abnormal sugar tolerance in all cases of "pseudo-diabetes."

NEW LIGHT ON PERIARTERITIS NODOSA

A. R. Rich of the Johns Hopkins Hospital, Baltimore, whose studies of allergy are well known, has produced evidence[3] that periarteritis nodosa may be a manifestation of the anaphylactic type of hypersensitivity. In the first of the cases which led him to this conclusion death had followed shortly after an attack of serum sickness. The opportunity to study the morbid histology of this condition has rarely occurred in the past: when serum was the only form of specific treatment available for pneumonia and meningitis, patients either died before serum sickness was due to develop, or recovered. Sulphonamide treatment now prolongs the lives of desperate cases in which serum has also been used, and serum sickness may have occurred before death. In several such cases vascular lesions indistinguishable from those of periarteritis nodosa were found

[1] Sylvest, O., *Acta med. scand.*, 1942, **110**, 183.
[2] Secher, K., ibid., p. 255.
[3] *Johns Hopk. Hosp. Bull.*, 1942, **71**, 123, 375.

in various organs post mortem. The naked-eye changes associated with this disease are not mentioned as having been observed, but the microscopical appearances of necrosis of the arterial wall with dense leucocytic infiltration in and around it are characteristic: haemorrhage around the vessel and the presence of eosinophils were sometimes observed and sometimes not. Of the 6 cases in which serum had been given, 5 had also had sulphonamides, but there were good reasons in 2 of them for regarding the rash and other manifestations as due to the serum and not to the drug, while in the sixth case serum alone could have been concerned. But the new argument does not stop here, because two other cases are described in which the same arterial lesions were found, and these patients had been treated with sulphonamides only: one had a typical drug fever with urticaria before death, the other a condition regarded as a febrile reaction to the drug but with no local manifestation other than a conjunctivitis. It therefore appears that sensitization to a sulphonamide compound may result in a vascular reaction of the same nature—that, in fact, serum sickness and drug fever, the clinical manifestations of which are certainly alike, have the same hitherto unrecognized basis. It is suggested that the drug may become linked with one of the plasma proteins, thus acting as a haptene, the body becoming sensitized to this compound; there is, indeed, evidence that this can occur. None of these patients exhibited any signs of periarteritis nodosa before the final illness, and successive biopsies were made in one case which fixed the time of development of the arterial lesions as coinciding with the clinical reaction of hypersensitivity.

Whether these findings can wholly bear the rather startling interpretation placed on them depends on whether the vascular lesion described can be considered truly specific. It is not inconceivable that necrotic and inflammatory arterial changes of this nature can arise in more than one way. This question brings strongly to mind the prolonged controversy which has been fought over the identity of the natural rheumatic lesion with those produced by the German school of allergists by means of foreign proteins to which the body had been sensitized. How far histological criteria can be depended upon in deciding aetiology is indeed a much broader question, on which there will be no general agreement. But Rich has performed a valuable service in opening a new line of investigation of an obscure disease. Any case of periarteritis nodosa should now be studied from this standpoint, and when the diagnosis has been made during life every effort should be made to determine whether a state of hypersensitivity exists to any identifiable agent.

INQUIRY INTO GROWING INCIDENCE OF JAUNDICE

The Medical Research Council, acting on a request from the Ministry of Health and in consultation with the War Office, has undertaken a special investigation into the causes of the present increased incidence of different forms of jaundice. The scheme is being generally directed by a committee of the Council under the chairmanship of Prof. L. J. Witts and with Dr. A. M. McFarlan as secretary. The headquarters of the investigation have been established in the Department of Pathology, University of Cambridge, by arrangement with Prof. H. R. Dean. The inquiries will be mainly made within a convenient radius of that centre, and cases occurring in military units as well as among the civilian population will be investigated. The team at Cambridge consists of five whole-time workers. The services of Dr. F. O. MacCallum as senior bacteriologist have been lent by the Wellcome Research Institution; Major Clifford Wilson (physician) and Captain J. A. R. Miles (clinical pathologist) have been seconded from the R.A.M.C.; and Dr. A. M. McFarlan (epidemiologist) and Dr. M. R. Pollock (assistant bacteriologist) have been attached from the Emergency Public Health Laboratory Service. In addition, Prof. S. P. Bedson is acting as general adviser on behalf of the committee. Prof. G. Hadfield has agreed to undertake the examination of histological material, and Prof. M. E. Delafield to collate epidemiological reports from various sources.

A TRACT FOR THE TIMES

The myth of the "machine man" dies hard. In spite of so much that has been discovered on the subject since the great industrial push following Dunkirk, the idea still persists among leaders of production drives and other people that the longer the hours worked the greater will be the output and that it will all be at the same qualitative level. Dr. H. M. Vernon has therefore done another good service by composing a tract entitled *Hours of Work and their Influence on Health and Efficiency*,[1] in which he assembles evidence to show the influence of working periods on the capacity of the worker. The evidence includes the reports of the Health of Munition Workers Committee in the last war, as well as the peacetime investigations of the Industrial Health Research Board, but mostly it consists of the results of inquiries on the industrial front since 1940. All this evidence points conclusively to the fact that excessive hours result in minor sickness, increased accident-proneness, and casual absence, and also in graver injuries to the worker. Readers of the *Journal* will recall the paper published less than a year ago (July 18, 1942) by Laidlaw and Macfarlane, based on observations particularly among women workers in Glasgow, which suggested that hours of work played an important part in the causation of pulmonary tuberculosis. Dr. Vernon is not content to rely solely on statistical argument, abundant as that is, but he points out fundamental psychological causes whereby the human factor will always in the end confound Pharaoh. There is, he says, an unconscious adjustment on the part of the worker to the hours he works. When the hours are long he gets into the habit of working at a slower pace. When these excessive hours are suddenly shortened the pace of the work for a time continues much as before, and production falls almost in direct proportion to the shortening of the hours; but presently, owing to the increase of vigour as a result of lighter work, the worker gradually and unconsciously speeds up—a process which continues for three or four months, and then attains equilibrium at a level substantially higher than that observed when longer hours were worked. Another factor in the present situation sometimes overlooked is that for women workers the toil does not usually cease when they leave the factory; shopping (never so exhausting as now) and household duties lay further exactions upon them. Dr. Vernon does not disguise his dislike of the regulation that women, unless they do 55 hours' work a week on business premises, must undertake fireguard duty. He suggests certain minimum hours for women and for girls and youths, while as for men he says that anything in excess of a 60-hour working week will bring no increase of production, and if the work is heavy, shorter hours than these are advisable.

The next session of the General Medical Council will open on Tuesday, May 24, at 2 o'clock, when the President, Mr. Herbert Lightfoot Eason, will take the chair.

[1] British Association for Labour Legislation, 21, Clareville Grove, S.W.7

THE ARMY BLOOD TRANSFUSION SERVICE

The Army Blood Transfusion Service is unique. No other army, Allied or Axis, has a distinct transfusion service with its own source of supply and its specially trained mobile resuscitation units. The headquarters of this Service were established six months before the war, and the plasma-drying plant, which was presented to it by the Silver Thimble Fund of the Women of India, was erected in 1941.

Scope of the Service

The Service now operates from Reading to Penzance in some 850 different centres. It has a staff of about 350, of whom more than three-quarters are women (either V.A.D.s or A.T.S.).

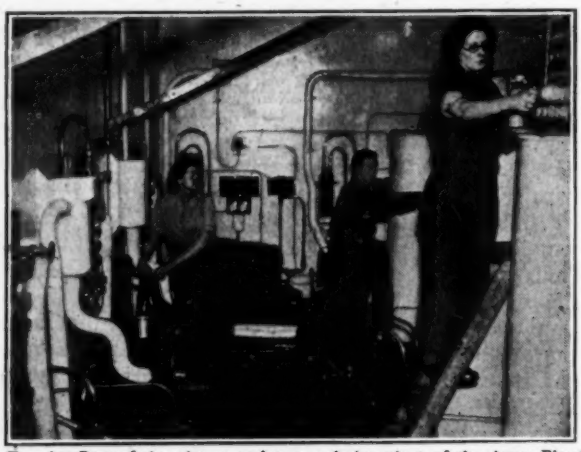


FIG. 1.—Part of the plasma and serum drying plant of the Army Blood Transfusion Service, showing the refrigerating and vacuum pumps. On the right one of the primary desiccators is being unloaded of its dried plasma.

FIG. 2.—Pooling and separation of the plasma and red cells. The nearest operator is pouring donated blood from the standard M.R.C. bottles into the container through the muslin gauze. The Alfa-Laval separator has two outlets—one for plasma (seen nearest the camera and filling the Winchester) and the other for the red cells on the other side.

The original technical staff was drawn largely from the laboratory staff of the Royal College of Surgeons and the Middlesex Hospital. The Service supplies, with transfusion fluids and equipment the whole British Army over-seas, from Iceland to the Cape of Good Hope and from Tunis to Burma. It supplies the basic requirements of the Forces at home and the civilian population in a Ministry of Health Region. It trains and equips units for service over-seas, and trains all ranks of the R.A.M.C. in resuscitation work. Since the beginning of the war blood has been taken from more than a quarter of a million donors. The 20 members of the staff of the Donor Registration and Publicity Department have to keep up to date the records of the three to four thousand donors who are bled each week. The bleeding is done by 15 mobile, self-contained, fully equipped teams, each consisting of a medical officer, 4 V.A.D.s, 2 A.T.S. drivers, and an A.T.S. orderly. The teams are detached, and quartered in the large towns, from which they "bleed" the surrounding area for a distance of 25 to 30 miles. Since 1939 two women doctors have between them bled more than 35,000 donors.

Transport of Transfusion Fluids

Whole blood is transported to any theatre of war within air distance of England, and in order to reduce shaking in transit the bottles are, by means of a special apparatus, filled to the top with glucose solution. Large insulated boxes are, by means of ice-inserts, kept at a temperature of 4° C. for a period of 8 hours. But most of the blood taken by the Service is separated, clarified, and filtered to make fluid plasma, which is sent out as such for military use at home and in temperate climates. That sent to the Army in tropical and subtropical regions is dried, and some twelve to fourteen hundred bottles come off the drying plant each week. Distilled water for reconstitution is sent with each bottle of dried plasma. Already some 56,000 pints of fluid plasma have been exported, and getting on for

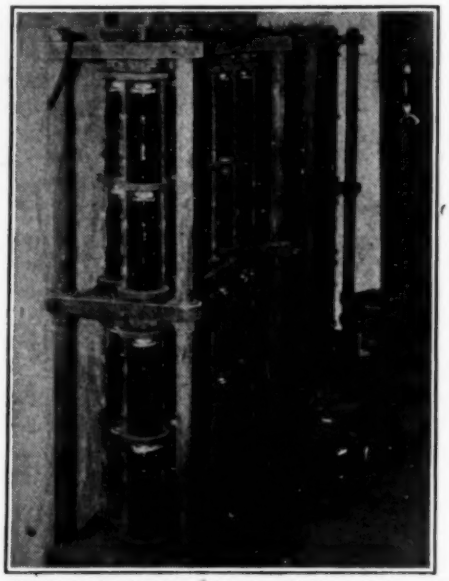


FIG. 3.—Bottles of liquid plasma in the spinning machine. The bottles in the photograph are actually spinning round at 1,000 revolutions per minute and stay so for 2½ hours at a temperature of −17° C. The effect is to form a vortex in each bottle, and when removed from the spinner each bottle contains what is in effect a tube of frozen plasma. Reconstitution with the appropriate quantity of sterile distilled water is complete in under 60 seconds.

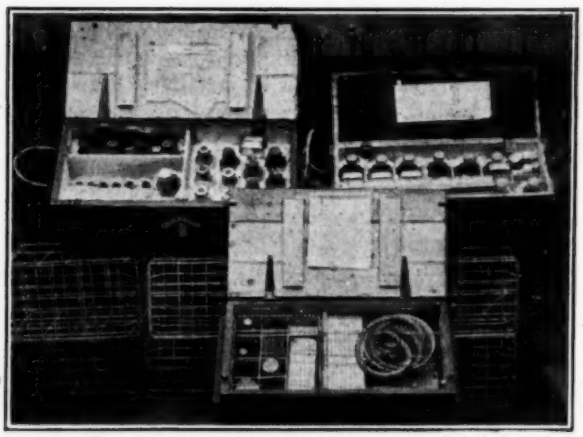


FIG. 4.—Three of the special boxes of the Army Blood Transfusion Service. *Bottom:* The field transfusion box—instruments, dressings, morphine, and drugs, tourniquets, etc., for the use of the medical staff. *Top left:* The field pattern box, containing sets for giving and taking blood; dried plasma and sterile distilled water, and glucose-saline. *Top right:* The specially adapted air-borne box containing instruments, dressings, drugs, wet plasma and dried plasma, and giving sets.

50,000 of dried plasma. Apparatus for administration assembled at the home depot is sent with all transfusion fluids, the apparatus being packed in tins, sterilized and ready for immediate use. The Service has also devised and issued special boxes of equipment to meet the transfusion needs of military hospitals, field ambulances, and troopships, as well as complete apparatus for full resuscitation by mobile field transfusion units and by airborne medical units. Another of the jobs of the depot in the South-West of England is the preparation of crystalloid solutions (glucose and saline) for all units at home and over-seas which are unable to prepare these themselves. The Service also supplies dried grouping serum.

Oversea Units

In every theatre of war over-seas the home service is linked to a base transfusion unit, which obtains from the headquarters at home the supplies required on the spot. These base transfusion units are also equipped for the production of crystalloid solutions, for the assembly of apparatus, for servicing and repair of refrigerators, and for obtaining blood locally. For example, the M.E.F. base unit has been able to maintain a service of whole blood to the 8th Army to supplement the dried plasma sent from home. Serviced by the base unit are mobile field transfusion units, fully equipped for the performance of resuscitation in the field. In North Africa these units have operated mainly in co-operation with field surgical units to form complete advance surgical centres with field ambulances, field dressing stations, and casualty clearing stations.

It has been said that the work of the doctor serving with the armed Forces has in this war been facilitated by three main innovations: (1) intravenous anaesthesia; (2) local application of sulphonamides to wounds; and (3) transfusion of blood and of plasma. Blood transfusion was, of course, used in the last war, but refrigeration and the use of fluid and dried plasma have revolutionized the application of blood transfusion in the field. That the fullest scope has been found for these new blood-transfusion techniques is due to the energy, wisdom, and brilliant organizing abilities of those who have built up this magnificent Service.

The photographs reproduced were taken by Mr. Norman K. Harrison of the "Topical" Press Agency Ltd.

CANCER RESEARCH

WORK IN PROGRESS

The annual meeting of governors of the Imperial Cancer Research Fund was held at the Royal College of Surgeons on April 29. The annual report, which was presented by Prof. H. R. Dean, chairman of the Council, described the continued work in various branches of research which has occupied the seven members of the scientific staff and the three visiting research workers during the last and previous years. The financial statement, presented by the treasurer, Sir Holburt Waring, showed the total receipts of the Fund during the last financial year to amount to £16,319, an increase of £1,561 as compared with the previous year. On the motion of Sir Hugh Lett, Viscount Halifax was re-elected president, and on the motion of Sir Alfred Webb-Johnson, Sir Holburt Waring was re-elected treasurer. Prof. H. M. Turnbull and Sir Hugh Lett were elected as "elected members" of Council for a period of three years, and the four "appointed members"—namely, Profs. H. R. Dean, H. W. Florey, C. A. Lovatt Evans, and John Beattie—were reappointed, also for three years.

In his report of the scientific work of the Fund[1] the director, Dr. W. E. Gye, F.R.S., observes that circumstances have been unfavourable to some kinds of research work—for example, to the study of viruses, for which large numbers of rabbits and fowls are needed. As small animals, such as mice and rats, are more easily reared and fed, work has been directed largely to investigating mammary cancer in mice, chemical carcinogenesis, the action of the hormones on the pituitary and the prostate, and to chemotherapy.

Carcinogenesis

Mr. H. G. Crabtree, M.Sc., has continued his work on the mode of action of chemical carcinogens, especially 3:4-benzpyrene. He has already shown that a series of halogen compounds can retard the development of skin tumours induced by pure chemical substances. The power to check such formation was parallel with the power to check glycolysis. Such compounds were also found to affect other chemical cell mechanisms, in particular those involving the sulphydryl group (−SH). Sulphur metabolism is now believed to play a part in the artificial induction of cancer. Apparently the general growth-inhibiting properties of many carcinogenic and non-carcinogenic hydrocarbons are related to the depletion of sulphur-containing amino-acids which co-operate in their elimination. It was therefore decided to make further investigations into the effect of sulphur metabolism on tumour induction. A brief description is given of experiments on the inhibiting effect of bromobenzene on skin tumours induced in mice by painting with benzpyrene. In one experiment an application of the former to the same area of skin painted by the latter increased the induction time for papilloma formation by about 50%. The organism detoxicates bromobenzene by combination with cysteine and subsequent elimination of the resulting mercapturate. From these and other experiments it is concluded that bromobenzene reacts with sulphydryl-containing tissue components. It appears that localized deficiency of sulphur is the determining factor in inhibition of tumour induction. It is thought that sulphur metabolism is intimately concerned in chemical carcinogenesis.

Dr. B. D. Pullinger's work suggests that trauma has a slight stimulating action in the production of experimental tumours. Trauma alone is, however, not enough, and the intervention of a specific carcinogen is apparently also necessary. This has a bearing upon the rare human instances of association between trauma and tumour.

Hormones

Dr. E. Vasquez-Lopez is working on the reactions of the pituitary body to prolonged treatment with oestrogens, implanted subcutaneously. It is found that under the influence of oestradiol benzoate and diethyl-stilboestrol the pars intermedia—not the anterior lobe—of the hamster becomes enlarged. The cells of the intermedia multiply and invade the posterior lobe, this being regarded as an increase in degree and extension of a normal process. The report points out that the hypophysis–oestrogen relationship has always been referred to the anterior lobe, and that these experiments at least serve as a reminder that the pars intermedia is a pituitary component which is functionally active. Similar overgrowth of the pars intermedia has been found in rabbits treated with thyroxine.

Tissue Culture

Dr. R. J. Ludford and Miss Hilda Barlow have been studying tissue culture of spontaneous tumours or tumours induced experimentally, previous observations having been made largely on the cells of transplanted tumours. It is found that, in general, carcinomas grown *in vitro* form compact sheets of cells, thus resembling normal epithelium. The fibroblastic type of growth is followed by sarcomas. Cultures of primary sarcomas contain many wandering cells as well as the malignant cells. In contrast there were few wandering cells in cultures of mammary carcinomas. No difficulty was experienced in distinguishing the morphology of sarcoma cells from that of fibroblasts, but when mammary gland of pregnant mice and spontaneous mammary carcinomas of high-cancer-strain mice were grown together in culture it was not possible to distinguish which cell was which by examination of the nuclear structure.

Fibroblasts grow prolifically when cultured with spontaneous mammary carcinomas of high-cancer-strain mice. A similar phenomenon was observed when fibroblasts were grown with non-malignant epithelial cells (growing embryonic kidney). In contrast, sarcoma cultures were found to inhibit the growth of fibroblasts. It is concluded that growing epithelial cells stimulate the growth of fibroblasts, and that the biological significance of this may be the need of epithelial cells *en masse* to have connective-tissue support.

In times like these it is much more difficult to carry on the kind of long-term research work which is needed in cancer than to turn attention to *ad hoc* war problems, and the profession will be glad to know that in spite of all these difficulties the Fund is continuing to give its valuable services to research; and medical men, too, will bear in mind that this work cannot be carried out without money.

[1] Fortieth Annual Report, 1942-1943, of the Imperial Cancer Research Fund, April, 1943.

Correspondence

Is Anaemia Prevalent?

SIR,—The absence of one of us on holiday has delayed this reply to your interesting leader (April 10, p. 449). The writer of that leader is under the impression that we "argue that there has been a general lowering of haemoglobin values in the population." In fact we agree with him that the evidence existing does not at present warrant any such conclusion. Though the haemoglobin level in the groups examined—of children as well as women—for example, by Davidson in Edinburgh and by ourselves in or near London, is, we think, disquieting, yet we hoped we had stressed the limited nature of our material. For example, we examined no men, few school children, and women in one factory only, not "in factories," as stated in the *Journal*, and, to quote our own words, "our investigation covers only a few sample groups, and the incidence of anaemia may well vary widely."

We have no evidence that would warrant the suggestion made in your article that "10% of the female population" have a haemoglobin level below 80%, and we have no means of assessing the number, nor, in spite of the absence of information on the limits of normal for women and children, do we think that many clinicians would accept 80% as the lower limit of normal for women.

We are glad that part of our aim is accomplished, for interest has been aroused in the subject, and (*pace* your leader writer) two official bodies at least—the Ministry of Health and the Medical Research Council—have initiated widespread haemoglobin surveys.

Your leader writer thinks that our town and country groups of students may not have been comparable. Actually, the choice of students for the London or country hospital was made by the medical school authorities on an educational basis, and no selection, such as he suggests, occurred. We had hoped we had made this clear. We owe an apology to the two hospitals concerned. By an oversight certain articles in the nurses' diet, practically pure carbohydrate and fat, were omitted from our computation. In London we were able to get details of these and they added 240 calories a day to the diet, and it is probable that an addition of the same order should be made to the country diet as given in our article. This was an error on our part, but does not invalidate our argument.

The aetiology of the existing anaemia certainly requires investigation, but that is too large a subject for discussion in this already overlong letter.—I am, etc.,

Royal Free Hospital, W.C.1. LUCY WILLS.

The Constitutional Factor

SIR,—I was fascinated to read your leader on the constitutional factor (April 24, p. 510). Prof. Bauer's contention appears to be that particular defects in the chromosomes are reflected in particular diseases in later life, the latter arising from foci of diminished resistance pre-ordained by the defective genes. This concept appears to agree substantially with my own theories, which I have applied particularly to the strain diseases, by which I mean those chronic constitutional diseases of increasing incidence in recent decades, like peptic ulcers, chronic rheumatism, diabetes, hyperthyroidism, arteriosclerosis, etc. Bauer's main conception, to quote your leader, "is that the genes act directly on the effector organ"—i.e., the organ through which the disease is expressed—"or indirectly through either the endocrine system or the nervous system, which can themselves collaborate."

The view expressed in my book, *Disease and the Social System*, is that, at any rate in the strain diseases, the primary factor is an innately vulnerable nervous system. In other words, I believe in a neuropathic aetiology in conditions other than those considered to be neurological or psychological in nature. I believe that this broad neuropathic diathesis is more specifically expressed in different varieties of dysfunction in the autonomic and endocrine systems. The specific pattern of the individual's disease is determined by the particular defect in his autonomic-endocrine liaison. An important observation in relation to the foregoing theory is the frequency with which, given the innate neuropathic diathesis, different members of the same family will produce different types of autonomic endocrine morbidity. One sees so often within the *milieu* of one family different varieties of the strain diseases.

It must be admitted that in some of the commonly associated strain diseases endocrine factors are not obvious according to the current tenets of clinical observation. Duodenal ulcer and chronic articular rheumatism are often found together in the same family. But I firmly believe, for reasons which space precludes me quoting here but which I have adduced in my book, that the endocrine factor in such diseases is of immense importance as well as in the deteriorating "abiotrophic" conditions like paralysis agitans.

But above all, at any rate in the commoner chronic diseases, the neuropathic factor is of first importance. The theory of a growing vulnerability of the nervous system may appear at first sight crude. In the last fifty years of improved hygiene and sanitation the only infective conditions showing a steady increase are the fevers affecting the nervous system. Is not this, perhaps, taken together with the increase in psychopathological conditions, a fact and an omen of great significance?

Apropos your last paragraph, I feel that, with all respect to the public health authorities, the art of preventive medicine is only being born, that it is a form of clinical medicine, and that it can only be elucidated by individual physicians for the benefit of individual patients, because it must consist so essentially in developing the patient's self-knowledge of his own innate and particular defects.—I am, etc.,

Bath. A. GUIRDHAM.

Direct Inguinal Hernia

SIR,—In the *Journal* of Feb. 11, 1939 (p. 263), Mr. W. G. Gill drew attention to a rare type of direct inguinal hernia and described three personal cases. I recently encountered a similar case at operation. Bearing in mind the apparent rarity of this condition, and being impressed by its very real existence as a variety of direct inguinal hernia, it seemed to be worth while to put the following on record.

The patient, a chief petty officer aged 46, presented himself for operation with a right-sided rupture of about 5 years' duration. He had first complained of a lump and of discomfort in the right groin following a strain while lifting a heavy weight when employed in a cordite factory. He consulted his doctor at the time and was fitted with a truss, which appeared to control the rupture adequately.

On examination there was a visible and palpable impulse on coughing and an appreciable enlargement of the right external ring. A pre-operative diagnosis of right inguinal hernia was made, which was thought to be most probably of the direct type. At operation a small hernial sac was encountered lying medial to the inferior epigastric vessels. It had a generous fatty covering almost amounting to an extraperitoneal lipoma, which had to be divided layer by layer before the sac could be completely displayed. (It closely resembled the typical femoral hernial sac.) It was about 1 in. long and had a narrow neck, which was found to be firmly engaged in a very obvious circular defect in the transversalis fascia. This had firm edges and would only admit the tip of the little finger. The opening was enlarged in order adequately to expose the neck of the sac, and on clearing its medial aspect the bladder was encountered and avoided. The neck was then ligated and the sac removed. The opening in the transversalis fascia was readily closed with interrupted sutures of No. 2 catgut. The cord was then replaced and no other repair of the posterior wall of the canal attempted.

He made an uninterrupted recovery, passing urine without difficulty from the beginning.

Although it is unreasonable to hazard a guess as to the causation of such a condition from the observation of a single case, my own feeling is that the sac develops as a direct sequel to an initial weakening of the transversalis fascia by a properitoneal lipoma as in the epigastric type of hernia (as suggested by W. H. Ogilvie in Maingot's *Post-graduate Surgery*, 1937).

My thanks are due to Surgeon Captain H. F. Briggs, R.N., for permission to publish the notes of this case.—I am, etc.,

M. R. EWING,
Surg. Lieut., R.N.V.R.

Treatment of Malignant Tertian Malaria

SIR,—The case of malignant tertian malaria recorded by Drs. W. L. Anderson and D. B. Bradshaw (April 24, p. 508) is of interest inasmuch as the man was apparently cured by quinine. The patient in the case recorded by Shute and myself in the *Lancet* (1942, **2,** 365) had, in point of fact, been taking regular doses of quinine, which suppressed the severe malarial symptoms without curing the disease, and it was not until quinine was discontinued that the diagnosis of malaria was possible.

In the case reported by Anderson and Bradshaw the small doses of quinine did not prevent the diagnosis of malignant tertian malaria being made, but it did suppress the severe clinical symptoms, and had quinine been stopped there is little doubt that these symptoms would have been much more severe. An important finding was the large number of gametocytes (crescents) present in the peripheral blood. This is evidence that the patient had been suffering from malaria for some considerable time, and that the quinine had failed to destroy the asexual parasites, because, had this been so, gametocytes could not have been so numerous. Mepacrine (atebrin) would have probably benefited the patient more than did the long-continued daily doses of quinine.

As regards the mepacrine treatment, when a severe case of malignant tertian is admitted to hospital it is the practice at Horton to treat the patient as follows: on the 1st day 0.6 g.; on the 2nd day 0.3 g.; on the 3rd and 4th days none; on the 5th, 6th, 7th, and 8th days 0.3 g. The reason for giving a reasonably large dose on the 1st day is that the parasites are subjected to drastic treatment, which will destroy most of the developing forms, either in the peripheral or internal circulation, particularly in the capillaries of the brain. The dose of 0.3 g. on the 2nd day will kill off most of the survivors of the previous day's treatment. The two-days rest followed by 0.3 g. for a further period of four days invariably cures the attack and possibly the disease as well.

Finally, a few small doses of plasmoquin 0.02 g. twice weekly for two or three weeks would have destroyed the sexual parasites (crescents) and so rendered the patient uninfective to anopheles mosquitoes.—I am, etc.,

Epsom. J. ELLIOT MURRAY, M.D.

The Calcium Intake in Pregnancy

SIR,—There seem to be three imperfections in the *Journal* for May 1. First, the "centrifuge tube for collection of leucocytes" described by Dr. M. A. Gohar (p. 542) is merely a copy of the tube introduced by Mindlin and Butler (*J. biol. Chem.*, 1938, **122,** 673) for separating the leucocyte-platelet layer of blood so that vitamin C may be estimated in it. Various workers have been using their method; we have been using tubes that contain about 3.5 ml. blood from which about 10 mg. of leucocyte-platelet layer are obtained, and the tubes are made by a laboratory glass-blower.

Secondly, your annotation on riboflavin and corneal vascularization (p. 545) seems to confuse the latter with circumcorneal injection. The two are obviously not synonymous. One connotes the formation of new vessels, the other the dilatation of existing vessels such as can be produced by rubbing the eyes. You say that the value of corneal vascularization as a sign of deficiency of riboflavin has recently been questioned in this country, quoting Scarborough. He confined his examination of the eye to circumcorneal injection, "but in no case was the slit-lamp used"; his observations therefore neither confirm nor refute the value of corneal vascularization as a sign of deficiency of riboflavin.

Thirdly, Dr. Robertson criticizes me (p. 550) for an alleged statement that "supplementary feeding of calcium . . . might prolong pregnancy and harden the foetal skull." My contribution to the meeting of the Scottish Group of the Nutrition Society was upon the evidence for nutritional deficiencies in pregnancy, and I was careful to state both sides of each question with which I dealt. Therefore, concerning deficiency of calcium in pregnancy, I spent some time on the well-known work on such deficiency, and on the other side included but one sentence upon possible dangers of calcium overdosage. My notes, I find, devoted 292 words to the work (which Dr. Robertson quotes) of Coons and the Toveruds, and on the other side only 15 words to the work of Abel, who observed (*Z. Ernähr.*, 1931, **1,** 366) that administration of a preparation of vitamin D prolonged the average duration of human pregnancies by about ten days, but who suggested that caution in the use of concentrates of vitamin D was necessary in case the bones of the infant's skull became too hard. I thought this work so unimportant that I omitted any reference to it in the written paper submitted for publication in the *Proceedings*. I have also stated in a letter to a colleague last March that I "completely agree with you in emphasizing the great value of a high calcium intake in pregnancy. However much chalk one ate one could not, I imagine, obtain Abel's results." I therefore consider that I anticipated the advice that Dr. Robertson now kindly gives me, in that I "stressed the well-established complications resulting from calcium deprivation rather than the unproven dangers of calcium overdosage."—I am, etc.,

Oxford. H. M. SINCLAIR.

Sulphaguanidine in Neonatal Gastro-enteritis

SIR,—Drs. C. Elaine Field and W. G. Wyllie (April 17, p. 486) say it is unfortunate that my clinical trial of sulphaguanidine in "infantile gastro-enteritis" was inadequately controlled, and apparently disagree with the statement in my paper (April 3, p. 410) that the greatly improved results achieved by its use did not justify this procedure. I would remind them that my paper was concerned only with epidemic gastro-enteritis in the newborn, which differs in some important respects from gastro-enteritis in older infants; also that controlled experiments involving serious risks can be justified only in experimental animals.

Research workers usually suspect the validity of inadequately controlled observations. Such a critical attitude is most necessary. I am not surprised, therefore, that my observations have failed to impress Drs. Field and Wyllie, as the value of sulphaguanidine was not proved in my series of cases by omitting it from the standard therapeutic regime in alternate cases. We all realize that clinical impressions, like inadequate controls, can be most misleading and are scientifically unsound, but I would assure my correspondents that all those with experience of neonatal gastro-enteritis who witnessed the investigation were so much impressed by the beneficial effect of sulphaguanidine that one would have been considered most inhuman to withhold it in any case.

My observations on sulphaguanidine therapy in neonatal gastro-enteritis were based on a relatively small series of cases, but the results were so promising that I thought it desirable to acquaint others, at this stage of the investigation, with its value in this disease, which causes so many deaths in maternity hospitals. I concluded my paper with the remark: "More experience with sulphaguanidine in neonatal gastro-enteritis is needed."

I was much interested in "the standardized plan of treatment" followed by Drs. Elaine Field, MacCarthy, and Wyllie in their careful study of gastro-enteritis in older infants (March 27, p. 371), but I was disappointed that they did not mention sulphaguanidine. I fully agree with their "further recommendations" (5–9) for prevention.—I am, etc.,

Edinburgh. J. L. HENDERSON.

Intolerance to Liver Extract in Pernicious Anaemia

SIR,—The importance of the article on intolerance to liver extract in pernicious anaemia (May 1, p. 539) is underlined by a patient of my own who died after an injection given on April 27. He was aged 83 and had been under treatment for pernicious anaemia for about five years. A year ago he suddenly became intolerant to his injection, and had one bad attack of dyspnoea and cyanosis and partial collapse relieved by adrenaline. After this there was a gap, during which ineffective attempts were made to get him to take sufficient liver extract by the mouth. Recently I decided to try again by injection, and instead of his usual dose of 5 c.cm. I gave a dose of 1 c.cm. Apart from slight skin irritation this was well tolerated, and four weeks later I gave a dose of 2 c.cm. In twenty minutes the patient was dead with acute respiratory failure, against which adrenaline and respiratory stimulants were useless. The article appeared four days too late.—I am, etc.,

Bristol. C. CHRISTOPHER MORGANS.

Treatment of Dysentery at Sea

SIR,—I have read with interest the letter of Flight Lieut. A. G. McManis and Major W. Gordon Rich on the treatment of dysentery at sea in your issue of Jan. 16. I fail to see, however, that treating dysentery on shore and at sea are so very different that injections of emetine hydrochloride have to be tried on the supposition that cases which do not clear up with ol. ric. and mist. bismuthi are due to amoebic infection.

In my experience there have always been a microscope and a few stains on board ship, and iodine and potassium iodide can be obtained from the dispensary for staining amoebae. Granted that cultures cannot be made, but fresh specimens of stools are easily obtained and stained. Bacillary dysentery can then be diagnosed on the typical cellular exudate found in this infection, and amoebic dysentery on the presence of the *Entamoeba histolytica.*

Treatment of bacillary dysentery has been by giving a mixture of sod. sulph. ʒij, mag. sulph. ʒij, aqua ʒj. An ounce was given immediately and ʒj hourly until the stools were watery, and the dosage was then reduced to three- and six-hourly administration until faeces reappeared. For the first 48 hours fluids only were given and then a typhoid diet (200–300 calories) but containing no milk.

I thought that by now bismuth had been proved to have no therapeutic action and have never used it. In cases where other antidiarrhoeic drugs have been given I have noticed that although the diarrhoea cleared up the patients suffered for many days with intense intestinal colic.

Many papers have so far appeared on the treatment of bacillary dysentery with sulphaguanidine. This drug has been advocated because it is only absorbed to a very small extent from the intestines. In this disease, however, the bowel is acutely inflamed, and one would not expect any absorption to occur. On the contrary, the capillary walls are so paralysed by the infection that fluid passes from the intestinal wall into the lumen. In consequence, I have seen no objection to treating severe cases of bacillary dysentery with sulphapyridine for the first 48 hours, especially since the mode of action of all these sulphonamide drugs is the same. Sulphapyridine also has the advantage of being more easily obtainable out here. Large doses have been given—12 g. per 24 hours—and then followed by salines. There have been no adverse effects, and the condition has quickly come under control.—I am, etc.,

Mhow, India. GRENVILLE MATHERS, Lieut., R.A.M.C.

Foot-and-Mouth Disease in Man

SIR,—The case of suspected foot-and-mouth disease described by Dr. K. MacLeod (March 6, p. 300) was also seen by me, and I can assure Dr. Monro (April 24, p. 520) that the condition was not consistent with Vincent's angina, the diagnosis of which is a relatively simple matter once diphtheria has been eliminated, being usually an ulcerative or membranous lesion, not vesicular, as was so marked in this instance. The two conditions are so fundamentally different that Dr. H. Dlugosz (Feb. 13, p. 189) has not considered it necessary to mention specifically Vincent's angina in his differential diagnosis.

The lesions in Dr. MacLeod's case were restricted to the mouth and lips, the tongue condition differing little from what is seen there in the bovine. The peculiar odour and the marked salivation also bore out the possibility of the case being actually a human infection with foot-and-mouth disease, but without laboratory proof one is naturally unwilling to be dogmatic on the matter. Inability to obtain a history of having drunk infected milk does not mean that this did not occur, for a child, acutely ill (who incidentally lives in a crofting community), is not likely to have very accurate recollections of what she ate or drank fourteen to twenty-one days before, and, as Dr. Dlugosz's article illustrates, ingestion of infected food is not absolutely necessary for infection although it is the most likely means.

The high resistance of man is well borne out in the case of the epidemic in this country where a dairy herd supplying approximately 20 gallons of raw milk to a county town did not produce a known case of the disease, in spite of the fact that doctors were on the look-out for such.—I am, etc.,

Dingwall, Ross-shire. J. LANDESS HORNE, M.D.

First Aid for Fractured Spine

SIR,—Following my letters on this subject in the *British Medical Journal* (Oct. 31, 1942, p. 529, and Dec. 19, 1942, p. 744) I submitted the matter to the executive committee of the British Orthopaedic Association. It was discussed at the last meeting held at the end of February, and the following considered opinion was given: "The executive committee decided to give authoritative support to the view that patients with suspected spinal fractures should be shifted and moved in such a way as not to be folded either backwards or forwards. The patient should be disturbed as little as possible, being transported 'as he lies.' From the point of view of the spinal injury, we are of the opinion that there is no indication for change of position from face to back or vice versa." I am sure all interested in this important matter will welcome this authoritative opinion on a difficult problem and will feel grateful for a clear and definite ruling. In view of this ruling the modified Clarke-Moir method of first-aid treatment for fractured spine or suspected fractured spine is being submitted to the B.R.C.S. and the Orders of St. John and St. Andrew for their consideration as to whether they can incorporate it in their teaching.—I am, etc.,

J. L. MOIR, M.B., Ch.B.,
Lt.-Col., Zone Medical Adviser, Anglesey Home Guard; County Medical and County Civil Defence Officer, Anglesey Branch, B.R.C.S.

Admissions to Mental Wards in Wartime

SIR,—I read with interest Dr. F. Hopkins's account of the decrease in admissions to mental observation wards during the war (March 20, p. 358). Dr. Hopkins's impressions are identical with my own, but I had hoped that he would be able to produce some significant statistics bearing on the subject. It is almost impossible to obtain any such data for the London area, because at various times during the war patients from the same area have necessarily been directed to different observation units, and there is some overlap in the areas served by different units. In the Liverpool area, however, where all cases were received in one unit, it should be possible to obtain population figures from the Ministry of Food authorities and to correlate these with the figures of admissions. If this were done the statistics published by Dr. Hopkins would acquire considerably more value.

At present it is not clear to me that the conclusions drawn from the published data have an adequate basis. Far too many variables are involved. It is, in my opinion, unwise to take the number of certified cases as a reliable guide. The considerations governing certification have altered somewhat owing to war conditions. Thus a patient with early senile dementia who might well have been cared for at home in peacetime may now be impossible to cope with owing to a risk of offences against the black-out regulations, or because he or she is too much of a liability under air-raid conditions, or because all the female relations are now at work and there is nobody at home to be responsible for the patient. Thus a considerably reduced population may be sending to hospital the same number of certifiable patients.

Proof of a reduction in the incidence of neuroses as a result of wartime conditions is of the utmost sociological significance for the reasons which Dr. Hopkins has indicated, and I hope that figures correlated with the population served will be published from his and other such units.—I am, etc.,

St. Mary Cray, Kent. BRIAN H. KIRMAN.

Scientific Freedom and Social Medicine

SIR,—Many of those who work in municipal hospitals will be in agreement with several of Dr. Geoffrey Bourne's general principles, but some of his points would not bear cross-examination. The picture produced of the municipal hospital has unduly sombre colours, while that of the voluntary system is somewhat too rosy, as no drawbacks are mentioned, though admitted by others. Municipal hospital staffs have considerable experience and appreciation of voluntary hospitals, but staffs of voluntary hospitals may only have a nodding acquaintance with municipal hospitals, which may be insufficient to draw definite conclusions.

In his criticism of the municipal hospital Dr. Bourne has focused attention upon the medical superintendent. There may

be medical superintendents who sometimes are not sufficiently tactful in carrying out some of their duties, just as there may be consultants who are sometimes forgetful of their hospital commitments. Similarly, examples could be quoted of doctors in any branch of medicine who sometimes interpret their duties inadequately; and cases could be stated, equally easily and fallaciously, to indicate that any particular system is essentially wrong. But all these matters call for adjustment in the individual, and isolated incidents cannot be the basis of a sound judgment.

There is no difficulty with regard to the formation of medical staff meetings in municipal hospitals so far as medical superintendents are concerned, for they are in favour of such a step. Senior staff have complete freedom of clinical action, but useful decisions could be taken at such meetings, and they would help to give consulting staff a closer interest and a more integral part in these hospitals with mutual benefit.

Both municipal and voluntary hospitals have their own advantages and disadvantages. Both exist solely for the care of the sick, and in their individual approach to the problem it is probable that neither has an all-round view, and each can learn something from the other. Voluntary hospitals have performed an inestimable service to the community, sometimes inadequately appreciated, but the rise of an alternative service has shown up certain points, arising from the fact that they have too much work to do. The enormous growth and improvement of municipal hospitals in the last twelve years and the confidence shown in them by practitioners and the public could hardly have been achieved if there were something radically wrong with their internal hospital medical arrangements.

The voluntary hospital system is an old and venerable tree that has borne much good fruit, but after so many years it is natural that there is some dead wood to cut away. In place of this should be grafted some of the vigorous growth of the new system, and together they will form a structure of unequalled service to the community.—I am, etc.,

Derby. R. G. COOKE.

SIR,—Dr. Geoffrey Bourne (April 17, p. 490) asks me three specific questions, to which I gladly reply.

1. I do believe in democracy in science as in everything else—that is, in real unrestricted freedom of speech and criticism.

2. I do not believe in placing autocratic power as regards medical affairs in the hands of any individual, be he medical superintendent or anyone else. Dr. Bourne in the letter just referred to states: "In plain fact the medical superintendent in council and municipal hospitals has absolute power as regards medical affairs as well as administratively." I can find no evidence for this statement. Rule 391 of the Hospitals and Medical Services Committee of the L.C.C. deals with the duties of a medical superintendent. It has twenty-eight sections, and the only one that could possibly be construed in the way Dr. Bourne suggests is Section VI, which reads as follows: "Organize and supervise the work of the medical staff of the hospital and be responsible for the due and punctual attendance upon the patients and for the giving of the requisite directions as to their treatment, nursing, and diet by the medical officers, and for the condition of the wards." It is difficult to understand how Dr. Bourne can say that this gives to the medical superintendent "absolute power as regards medical affairs as well as administratively." Rightly or wrongly it has been our traditional method to evolve from one system to a better by gradual steps and stages. In a sense, therefore, the master of a Poor-Law workhouse may be looked upon as the remote ancestor of the medical superintendent of a council hospital. But the absolute power of the medical superintendent is gone, although he is still administratively in charge of the institution.

3. I believe that medical committees should be instituted in every hospital, and that they should have local freedom to elect their own chairmen and officers, and that machinery should be devised, where it does not already exist, so that these committees may convey their opinions directly to the body in control of the hospitals in which they work.

Dr. Bourne goes on to say, referring to myself: "If his official position makes it impossible for him to reply, I suggest that he ascertains the answers from his lay authority with their permission to make them public." Whether this foolish remark arises from ignorance or prejudice I do not know. I am a member of the honorary staff of a voluntary hospital and a part-time surgeon under the E.M.S. scheme. I have criticized the voluntary hospitals and the E.M.S. scheme freely without let or hindrance and shall continue to do so if necessary. I am also a member of the L.C.C. and chairman of its Hospitals and Medical Services Committee. The L.C.C. is a democratic organization, every member of which is free to express his views, although, obviously, where decisions have to be made it is the view of the majority that prevails. I do not know to which of these three "lay authorities" (or any other) Dr. Bourne refers.—I am, etc.,

Northwood. SOMERVILLE HASTINGS.

A National Medical Service

SIR,—We do not deny that the present medical services are far from perfect, and no member of the profession would impede any alterations calculated to achieve the maximum possible efficiency. It is the stated policy of the present Government (elected in 1935) to institute a unified medical service; but approximately one-seventh of the total population of these islands, selected by medical boards from a selected age group, require the attentions (under a unified medical service) of about a quarter of the total profession (*Lancet*, April 10, 1943, p. 469). The success of any medical service must depend on the active and willing co-operation both of the lay public and of the profession. But at present the most virile section of the public (lay and professional) is disfranchised, and has not been, nor can be effectively, consulted in wartime.

We suggest, therefore, that the present time is inappropriate for the Government to foist a new medical service upon a public, unsuspecting, and gagged by the bureaucratic restrictions necessary to the successful conclusion of the war.—We are, etc.,

DOUGLAS ROBERTSON.
G. O. RICHARDSON.
G. A. SMART.

Kenny Treatment of Poliomyelitis

SIR,—My attention has been drawn to the leading article in the *Journal* of Nov. 28, 1942 (p. 639), entitled, "Kenny Treatment of Poliomyelitis." In reference to this matter I consider it necessary to correct certain statements contained in this article.

In the first instance my contribution to medicine is not a reformed treatment for recognized symptoms but a new concept of the symptoms. With regard to this I would refer you to the bulletin read at the staff meeting of the Mayo Clinic, Aug. 12, 1942, and presented by Dr. Frank Krusen, professor of physical therapy, Mayo Clinic, Rochester. Dr. Krusen admits that when he heard my theory with regard to these symptoms he thought I was unbalanced, but when I invited him to come to Minneapolis and see for himself he, with the heart and mind of the true physician, readily accepted the invitation, and, subsequently, requested to be enrolled in the first physicians' class to be held under the auspices of the Department of Physical Therapy and Orthopaedic Surgery, School of Medicine, University of Minnesota, during which time the concept was presented to him and the treatment for this new concept demonstrated and the results shown. Dr. Krusen's report reads: "Her ideas are original and she should be given full credit for having developed a new and extremely interesting concept of the symptoms of early poliomyelitis and the proper management of these symptoms. The Kenny method merits the close scrutiny of every physician."

Your article makes reference to the main points of my treatment in the acute stage. I would respectfully draw your attention to the fact that patients were not admitted to Queen Mary's Hospital in the acute stage. The patients treated in this institution were transferred from the infectious diseases hospital after the acute stage was over, and in a great many instances were not admitted to Wards A7 and 8, where the work was being carried out, but were hospitalized in E3 and 4 until the London County Council inquired of the parents if they were willing that their child should be submitted to the new type of treatment. This took quite a while. The parents generally waited to consult the parents of the children who were already receiving treatment, after which they would give their consent. By this time in quite a number of the cases the early symptoms had accomplished their deadly work. I attempted to explain this to the committee, and also that spasm was a damaging feature, but the committee were non-receptive. The reply I received was, "Spasm? What do you mean? This is a new one on us." This remark was passed by Mr. Fairbanks, and nothing further materialized. This committee was formed in Nov., 1937, during my absence in Australia.

I returned to England in April, 1938, and met the committee twice—in April, 1938, and May, 1938. The report referred to was presented in June, 1938. I left Queen Mary's Hospital the following month and returned to Australia. The conferences held at Queen Mary's Hospital by the committee during the two afternoons of their visit in April and May and their final conference in June, before the presentation of their report, were held *in camera*. I was not invited to be present.

I would also like to state that you are mistaken if you think that I was well pleased with my reception by the L.C.C. I was well pleased with the earnest endeavours of the technicians to grasp the technique taught them, which prevented deformities despite the abandonment of the paramount principle of orthodox procedures—immobilization. I was bitterly disappointed at the refusal of the L.C.C. to allow the Kodak Company to reproduce the film picture of my work at Carshalton, for which I myself was willing to pay in order that I might take it back to Australia. Sir Earle Page, Minister of Health of the Commonwealth of Australia, had made the request to me. I also noticed that you make reference to a report submitted by Dr. Starr, given to the Minister of Health, New South Wales. I think it is well known that I had dissociated myself from this work with Dr. Starr long before this report was published.

I notice that you have made no mention of the report submitted by Drs. Forster and Price of Melbourne, Australia, in which they state: "(1) Miss Kenny lays great stress on the condition of muscle spasm, its prevention and cure. We think her views on this subject require careful consideration. (2) Stiffness is a troublesome feature in the cases treated by the orthodox methods and entirely absent in the cases treated under Miss Kenny's supervision. The reason why should be earnestly sought. (3) Expected deformities from muscle imbalance did not occur, but did occur in our own splinted patients we ourselves treated or received from other surgeons. This calls for comment."

You also quote: "In December, 1941, the *Journal of the American Medical Association* made this cautious statement: 'Adoption of the Kenny technique represents an elaboration of well-recognized principles,' and, 'If there is any revolutionary element it consists in its abandonment of early rigid splinting and the adoption of continuous and meticulous hydrotherapy and physical therapy to maintain the function of muscles, at the same time producing increased comfort to the patient.'" This statement appeared (like your own) in the editorial, and was presented by one whom I had never met or at that time seen. You omitted to quote the statement made in the issue of this journal for June 7, presented by the professors of physical therapy and orthopaedic surgery, University of Minnesota, who had observed the work and made their report from observation, reading: "According to this concept the cardinal symptoms of infantile paralysis are to use her terms muscle spasm, muscle incoordination, and mental alienation. This is opposed to the usual concept, where the cardinal symptom is flaccid paralysis without muscle spasm or incoordination. The presence of spasm has been demonstrated in 100% of the cases." This report further refers to this matter and states the concept is original and unconventional.

You also state: "In the textbooks it is recognized that in poliomyelitis there is gross disorganization of muscular function with overaction (which is another way of saying spasm) of unparalysed muscles." Again, "That mental alienation is no new idea is well shown by the following sentences from Jones and Lovett: 'Muscle training is fundamentally an attempt to restore a cerebral motor impulse to a muscle, an impulse which has been either impaired or lost during the acute stage of poliomyelitis.'" It is made clear in this presentation that the writer of this article is not familiar with the Kenny concept. He or she refers to overaction of unparalysed muscles. These are in orthodoxy supposed to be normal muscles contracting and pulling the skeleton into deformity. These muscles are in reality the sick muscles, not the non-paralysed one. They are the muscles that require careful nursing. The pain, spasm, and shortening causing the deformities are centred in these muscles, and the alienation of the opponents is caused through this condition, not as stated by the writer in his paper.

The writer further states in reference to Lovett and Jones: "They knew all about incoordination, overaction or spasm of muscles, cerebral impulses, and the scrupulous care required for their protection and re-education." The presentation of the muscle condition as accepted by Lovett and Jones is reproduced in *Public Health Bulletin*, No. 242, U.S.A. Public Health Headquarters, by the pupils of Dr. Lovett, and reads: "The muscle itself is never affected by the disease but is comparable to an automobile with its battery stolen. The muscle is loose and flaccid and hangs like a hammock between its points of origin and insertion." This is the Lovett-Jones concept for which splinting was applied to prevent the strong supposedly normal muscles from pulling the weak, flaccid muscles. Nurses were warned that to leave a splint off the part and the supposedly flaccid muscles unsupported even for a short time would mean prolonging treatments for months.

These supposedly flaccid and paralysed muscles were the muscles referred to in the report of Dr. Alfred Deacon, orthopaedic surgeon, Children's Hospital, Winnipeg, Canada, which appeared in the *Canadian Public Health Journal*, August, 1941, reading: "We were astounded to see Miss Kenny restore what we had recorded as completely flaccid and paralysed muscles to full function in the course of a few minutes by restoring mental awareness." This was not done by directing the patient's mind to the part. The motor pattern had to be restored first. The patient is not asked to think. If they could think there would be no necessity to tell them to do so. The procedure reaches further than that. The concept can be best explained in the language of the scientist and research worker, Dr. Hipps, who wrote: "You have found symptoms and visible findings in this disease, infantile paralysis, that we had no idea existed. You have demonstrated to me a disease I did not know existed."

You are very much mistaken if you think the American workers are giving extravagant credit to this presentation or that it is old ideas with new names. There have been upward of 300 doctors attending the classes at the University of Minnesota, all of whom have practising technicians also trained in Minneapolis from the United States of America, Cuba, and Canada. Many have film pictures of the results and that of their orthodox patients, showing 60% more recoveries in the patients treated by the method evolved for the new concept, which has been acknowledged by the observers to be diametrically opposed to the orthodox concept.

The author of the article in your *Journal* refers to Jones and Lovett as the orthodox authorities on this disease. The two physicians referred to in this letter—Drs. Krusen, Mayo Clinic, and Pohl, Minneapolis—were trained in this school, and upon my arrival in Minnesota, U.S.A., were religiously practising their technique. Both thought I was unbalanced mentally when I presented my concept, and both, being true physicians, now acknowledge their mistake. The investigation of my work and theories was carried out at the suggestion and under the direction of Dr. Wallace Cole, professor of orthopaedic surgery, University of Minnesota, and was assisted by Dr. Miland Knapp, professor of physical therapy. I would gladly welcome the author of your article to Minneapolis as representative in the interests of humanity and the well-being of the people of England. I think this would be a laudable undertaking.

I have requested the Bruce Publishing Company to forward to you the book entitled: *The Kenny Concept of Infantile Paralysis and its Treatment*. You will see in the foreword, written by Dr. Frank Ober, professor of orthopaedic surgery, Harvard University, and president of the American Orthopedic Association, that: "Sister Kenny puts great emphasis on muscle spasm in the affected muscles. One does not find mention of muscle spasm in textbooks, but there is no doubt that it is present, and is a great factor in the production of early deformities unless relieved. Mental alienation of muscles is another term used in the book, and does occur even in other conditions in which there is no paralysis." Dr. Ober was a colleague and pupil of the Jones and Lovett you refer to. This evidence, presented from indisputable authority, will go far to explain the reason why deformities did not occur in the patients treated by this method under my supervision at Queen Mary's Hospital, Carshalton, and every other institution that adheres to the method when immobilization, the paramount principle of orthodox procedures, to prevent this condition was abandoned.

You will learn from the above observation and statement that my concept is not a new term for old ideas. It is the muscle affected that is in spasm and the non-affected one that is alienated. Therefore, the explanation given by Drs. Cole and Knapp two years ago is supported to-day by Dr. Frank Ober and the hundreds of others that have come to listen and learn. It is explained in the presentation of Dr. Frank Krusen, Mayo Clinic, Rochester, and already quoted in this letter. I would gladly welcome a doctor and technician from Queen Mary's Hospital to finish their training, also from any of the Australian clinics that are equipped with a partially trained staff. Units are being enrolled from the South American Republics, Cuba, Canada, and China. The University of Minnesota would gladly share this message of healing with the rest of the world.—I am, etc.,

Minneapolis. ELIZABETH KENNY.

Staffing of Miniature Mass Radiography Units

SIR,—With reference to Dr. Norman Henderson's letter (April 17, p. 488), reduction in radiographic exposure time when employing mobile power unit is due to a variety of reasons. A specific input voltage at a high frequency permits precise calculation of iron and copper, and, more particularly, allows reduction of core and windings in the design and construction of the high-tension *x*-ray transformer. Portability of transformer, therefore, is the logical reason I have specified, in certain instances, a high cycle input in self-contained mobile *x*-ray units (*Brit. J. Radiol.*, 1934, **7**, No. 79).

I would not advocate the adoption of non-standard frequency in any contemplated wide use of mobile miniature radiographic units. Standardization must be preserved throughout the scheme whether the apparatus be used on the public supply mains or

the mobile generator. Improvement in radiographic exposure time is due primarily to distortion of wave-form in the alternator on load, resulting in a "peaky" rather than true sine wave. Under certain conditions an induction coil effect is produced which varies according to the regulation of the transformer.

I apologize for dogmatizing on a technical subject through the columns of a medical journal.—I am, etc.,

London, S.W.1. H. T. FERRIER.

Maintenance of the Dependants of Tuberculous Patients

SIR,—The Committee on Tuberculosis in Wartime in their report (M.R.C. Special Report Series 246, 1942) made the suggestion, now adopted by the Minister of Health (Memo. 266T, April, 1943), that financial aid from public funds should be afforded to the dependants of tuberculous patients during the period of the breadwinner's invalidity. It is now more than 40 years since this suggestion was made to the Corporation of the City of Manchester by their then medical officer of health, the late Dr. James Niven. A passing reference to the incident is recorded in his *Observations on the History of Public Health Effort in Manchester* (Jno. Heywood, 1923).—I am, etc.,

Ministry of Health, Whitehall, S.W.1. J. R. HUTCHINSON.

Obituary

C. LAMBRINUDI, F.R.C.S.

Orthopaedic Surgeon to Guy's Hospital

We regret to announce the sudden death on April 21, at Mayfield, of Mr. C. Lambrinudi, orthopaedic surgeon to Guy's Hospital and consulting orthopaedic surgeon to Queen Mary's Hospital, Carshalton, who had made many valuable contributions to his branch of practice.

Constantine Lambrinudi was born in 1890 and entered Guy's from Christ's College, Cambridge, in 1910. His medical studies were interrupted in 1912-13 by two periods of service during the war in the Balkans. He qualified M.R.C.S., L.R.C.P. in 1914, and was in turn assistant house-surgeon to Mr. C. H. Fagge, out-patient officer, and house-surgeon to Sir Alfred Fripp. During the last war he served again with the Greek Army and later as a medical officer in the Royal Flying Corps. After working for a year as part-time demonstrator of physiology at Guy's and surgical registrar at the Royal Free Hospital he took the F.R.C.S. in 1921, and was chief clinical assistant and registrar in the orthopaedic department at Guys from 1923 to 1928, when he was appointed assistant orthopaedic surgeon; on the death of Mr. Trethowan in 1934 he became orthopaedic surgeon in charge of the department. Lambrinudi was honorary secretary of the Orthopaedic Section of the Royal Society of Medicine in 1933-5, and served on the Council of the British Orthopaedic Association. He described a new operation for the correction of drop-foot in the *British Journal of Surgery* in 1927, and wrote on the "Use and Abuse of Toes" in the *Clinical Journal*. At the Annual Meeting of the British Medical Association in 1934 he opened a discussion in the Section of Orthopaedics on adolescent and senile kyphosis, and this paper appeared in the *British Medical Journal* of Nov. 3, 1934.

A tribute published in *Guy's Hospital Gazette* recalls the fresh and unbiased outlook which Lambrinudi brought to every problem that he tackled.

"The result was not only the gift to orthopaedics of a considerable number and variety of real advances, which have already been generally accepted, but also original and stimulating thought on nearly every subject which he touched. He had also that other quality of greatness—simplicity in ideas. . . . He held that the chief function of the specialist was to explore the uncharted, or only partially understood, regions of medicine, to elucidate and simplify them, and return the knowledge so obtained to medicine as a whole. He held that there were no principles of orthopaedics as opposed to principles of medicine. There were only principles of medicine, which in orthopaedics should be applied to the restoration and maintenance of the function of locomotion, using the term in its widest sense. Always he laid stress on function as the only true guide in treatment, and not only function of the body as a whole, but also its proper functioning in relation to its social environment. It was for this reason that he attached so much importance to the almoner's department, and it was for the same reason that he instituted the employment of the visiting masseuse in connexion with hospital practice—an innovation in which Guy's has led the field."

Mr. FRANK ST. J. STEADMAN, senior dental surgeon to the Royal Dental Hospital, London, died suddenly on April 11, aged 63. He was a student at Charing Cross Hospital and took the L.D.S. in 1902, following this with the M.R.C.S., L.R.C.P. in 1907, and the D.P.H. in 1911. Devoting himself to the practice and teaching of dental surgery, he was appointed to the staff of the Metropolitan Hospital and the Belgrave Hospital for Children, and lectured in histology and practical dentistry at the National Dental Hospital. During the last war Mr. Steadman served over-seas in the R.A.M.C.(T.A.), and reached the rank of major when holding the administrative post of D.A.D.M.S., 20th Corps. After returning to civilian life he became lecturer in dental diseases in childhood, on local anaesthesia, on bacteriology, and on oral surgery, at the Royal Dental Hospital. He also examined in dental surgery for the Universities of London and Bristol. In 1937 he was president of the British Society for the Study of Orthodontics, and in 1939 president of the Odontological Section of the Royal Society of Medicine.

Dr. ERNEST CHARLES JENNINGS died at Newton Abbot on March 13 as the result of a motor accident. He was 65 years of age. He studied medicine in Dublin, and qualified at the Royal College of Surgeons in 1904. He held resident appointments in England, and went to Australia in 1911, where he did much pioneer work in establishing medical services in the remote districts. He practised in Queensland and New South Wales. In 1928 he returned to England, and took up practice at Bracknell. Two years ago he retired and went to Devonshire. Dr. Jennings was a member of the B.M.A., and always took an interest in all medical work until the time of his death. His cheerful and lively personality endeared him to all with whom he came in contact, and he will be much missed by his colleagues and friends. His wife survives him.

The Services

Capt. (temp. Major) G. H. G. Hope, R.A.M.C., has been awarded the M.C. in recognition of gallant and distinguished services in the Middle East; and Capts. (temp. Majors) L. E. C. Davies and J. R. Kellett, R.A.M.C., have been appointed M.B.E. (Military Division) in recognition of gallant and distinguished services in the South-West Pacific.

CASUALTIES IN THE MEDICAL SERVICES

Wounded.—Capt. N. J. H. Davidson, R.A.M.C.

Missing since the Fall of Singapore, now Officially Reported Interned in Malaya.—Dr. M. A. X. Cocheme, Colonial Medical Service.

Missing, Presumed Killed.—Surg. Lieut. Philip Alan Rees, R.N.V.R.

DEATHS IN THE SERVICES

Surg. Rear-Admiral HERBERT REGINALD HARRY DENNY, C.B., R.N. (ret.), died on April 20 as the result of a motor accident at Plymouth, aged 66. He was educated at Guy's Hospital, and, having obtained the Conjoint Board medical qualification in 1899, joined the Royal Navy in the following year. He served throughout the war of 1914-18 in the Grand Fleet and subsequently became S.M.O. of the Royal Naval Hospital, Cape of Good Hope. In 1931 he was promoted to be rear-admiral and in 1935 he was appointed honorary surgeon to the King. He received the C.B. (Mil.) in 1932 for his long and distinguished services. After his retirement in 1935 he resided at Plymouth, and he was engaged in war work at the time of his fatal accident.

Lieut.-Col. WALTER ROTHNEY BATTYE, D.S.O., I.M.S. (ret.), died, aged 69, at Parkstone on April 27 after a long illness. He was a highly qualified and able officer who had a varied career. He obtained an entrance exhibition and gained medals at University College, London, and took the B.Sc.Lond. in 1898, with honours in zoology, the M.B., B.S. in 1897, the M.S. in 1908, and the F.R.C.S. in 1909. He entered the I.M.S. in 1898, gaining the second place, and won the Parkes Memorial Medal at Netley. After serving with Indian cavalry regiments in India he saw active service in China during the Boxer rebellion in 1900. Three years later he entered the civil branch of the I.M.S. in the coveted political department, served during the next ten years in Persia and in Rajputana, and became

chief medical officer, Central India, residency surgeon, Indore, and superintendent of the King Edward Hospital and Medical School, where his exceptional qualifications found scope. During the war of 1914-18 he served in Egypt, and in the Dardanelles until the evacuation, and was mentioned in dispatches four times, awarded the D.S.O., and made a Chevalier of the Legion of Honour. After duty in France he was promoted to be D.A.D.M.S. at Baghdad, and subsequently became Secretary for Health and Director of Civil Medical Services, Mesopotamia, in 1919. After this varied and distinguished career he retired in 1929, but served further as medical superintendent of King George V Merchant Seamen's Memorial Hospital, Malta. He was the author of papers on plague, spinal anaesthesia, the treatment of Anzac wounded, and on the climate and health of Meshed, Persia. His life's work well illustrates the great opportunities the Indian Medical Service in its palmiest days offered to men of his ability and energy, of which he took the fullest advantage.

Lieut.-Col. ALEXANDER CLIFTON CRAIGHEAD, I.M.S., died at sea through enemy action in March, 1943, aged 54. He studied medicine at St. Andrews University and passed the final M.B., Ch.B. examination in 1914. He became a temporary lieutenant, R.A.M.C., during the last war in 1916 and obtained a permanent commission as captain in the I.M.S. in 1919. In 1925 he took the D.T.M.&H. with distinction, and gained the Duncan medal. He served in the Indian Research Department and became assistant director, Central Research Institute, Kasauli, and was joint author of an investigation into the value of an etherized vaccine.

Universities and Colleges

UNIVERSITY OF OXFORD

In Congregation on May 6 the honorary degree of D.Sc. was conferred on Dr. Joseph Trueta, formerly Director of Surgery in the University of Catalonia, who came to England in 1939 and in 1940 was appointed by the Minister of Health to an advisory post at the Wingfield-Morris Orthopaedic Hospital, Headingley.

UNIVERSITY OF CAMBRIDGE

Albert Charles Chibnall, Sc.D., F.R.S., of Clare College has been elected Dunn Professor of Biochemistry from Oct. 1, 1943, in succession to Sir Frederick Gowland Hopkins. Dr. Chibnall has held the chair of biochemistry at the Imperial College of Science and Technology, London, since 1936.

At a Congregation held on April 30 the following medical degrees were conferred:

M.D.—(by proxy) J. W. Parks, T. E. Jones-Davies, (in person) H. J. Wallace.
M.B., B.CHIR.—(by proxy) L. G. Kingdom, B. Pownall, J. Lomas.

Titles of the degrees of M.B., B.Chir. were conferred by diploma in March on E. Tylden of Girton College, and in April on H. C. Mason of Newnham College.

The Professor of Anatomy gives notice that applications for the Marmaduke Sheild scholarship in human anatomy are to be sent to the Registry by May 20 in the present term.

UNIVERSITY OF LONDON

The membership of the following medical representatives of Convocation has been continued until May, 1944: *Medicine*, Sir E. Graham-Little and Major-General P. H. Mitchiner; *Science*, Prof. M. Greenwood, D.Sc., F.R.S.

The following candidates have been approved at the examination indicated:

POSTGRADUATE DIPLOMA IN PSYCHOLOGICAL MEDICINE.—[1]G. H. H. Benham.
Part I: A. H. D. Hunter, A. W. MacLeod.

[1] With Mental Diseases (Psychiatry).

UNIVERSITY OF GLASGOW

The degree of Doctor of Medicine (M.D.) with commendation was conferred by diploma upon James Stewart, M.B., Ch.B., on April 17.

ROYAL COLLEGE OF PHYSICIANS OF LONDON

At a meeting of the College, held on April 29, with Lord Moran, President, in the chair, the following were elected Fellows:

C. R. Burns, M.D. (New Zealand); B. Maclean, M.D. (Newcastle, Staffs); Janet K. Aitken, M.D. (London); H. L. Wilson, M.D. (London); H. Joules, M.D. (London); L. Minski, M.D. (Sutton, Surrey); A. G. Watkins, M.D. (Cardiff); Brig. F. M. Lipscomb; Major-Gen. Sir P. S. Tomlinson, C.B., D.S.O.; C. T. Andrews, M.D. (Truro); G. A. Elliott, M.D. (Capetown); Reginald Ellis, M.D. (Manchester); M. Kremer, M.D. (London); G. D. Kersley, M.D. (Bath); D. U. Owen, M.D. (Liverpool); R. Bodley Scott, D.M. (London); W. E. Snell, M.D. (London); F. S. Fowweather, M.D. (Leeds); R. H. Parry, M.D. (Bristol); K. Robson, M.B. (London); J. L. Lovibond, M.B. (London); P. J. Kerley, M.D. (London); Denis J. Williams, M.D. (London); W. Ritchie Russell, M.D. (Edinburgh); J. W. Bigger, M.D. (Dublin); Noah Morris, M.D. (Glasgow); J. E. Gordon, M.D. (Harvard); James M. Mackintosh, M.D. (Glasgow); Charles McNeil, M.D. (Edinburgh); L. C. Montgomery, M.D. (Montreal).

Dr. J. L. Livingstone was elected an Examiner in Pathology.

The following candidates, having satisfied the Censors' Board, were admitted Members:

J. L. Bates, M.B., J. L. D'Silva, M.B., T. S. Eddie, M.B., L. A. Hawkins, M.B., J. C. Herapath, M.B., A. W. J. Houghton, M.B., Major R. P. Howard, M.D., R.C.A.M.C., J. S. Lawrence, M.D., R. Marshall, M.D., Surg. Lieut.-Cmdr. G. J. Murray, M.D., R.N.V.R., Sheila M. Newstead, M.B., Capt. O. R. L. L. Plunkett, L.R.C.P., R.A.M.C., K. O. Rawlings, M.B., F. Ridehalgh, M.B., Sheila P. V. Sherlock, M.B., J. V. Sparks, L.R.C.P., J. R. Tasker, M.B., W. G. Tillmann, M.B., A. W. W. B. Woods, B.M.

Licences to practise were conferred upon the following 238 candidates (including 30 women) who had passed the Final Examination in Medicine, Surgery, and Midwifery of the Conjoint Board, and have complied with the necessary by-laws:

D. G. Abrahams, N. Ambache, I. B. Amin, R. E. Arango, E. L. Arnold, D. C. Artingstall, Janet F. Atkins, D. R. F. Atkinson, J. Attenborough, G. B. Badock, R. A. J. Baily, Ursula M. Baines, E. H. D'A. Banister, E. A. Barker, G. D. G. Barnes, Kathleen B. Bentley, D. H. B. Bevan-Jones, A. I. Black, Katherine M. Blacklock, W. R. Bodenham, G. L. W. Bonney, Margaret E. M. Boulton, B. T. Bowen, J. C. Brace, W. Brand, A. R. Brandreth, E. Brenman, M. Bresler, D. L. Bridgewater, J. G. Brown, M. M. Brown, P. F. E. Brown, E. A. Burkitt, J. D. Burton, R. J. Bush, R. J. Cairns, R. W. L. Calderwood, Janet Cameron, E. F. Carr, S. Chakravorti, G. I. Clarke, Peggy C. Cole, E. Consterdine, M. Conway, M. A. Cooke, J. C. Cooper, G. N. C. Crawford, D. G. Crawshaw, Alexandrina Currie, Vera J. Cutler, A. G. Davies, T. D. L. Davies, P. R. H. Davis, M. V. Doherty, G. H. P. Drake, P. D. A. Durham, W. H. Dyson, J. R. Elliott, J. W. Ellis, Norah C. Elphinstone, A. Elton, D. W. Evans, E. P. Evans, J. W. Evans, D. A. T. Farrar, L. Feldman, T. R. Fink, H. H. Firth, H. J. W. Fisher, C. J. Foord, Edith V. Foyle, R. S. Francis, H. J. Furstenheim, Sylvia B. Gadsden, M. B. Garnett, H. N. H. Genese, G. H. Gilford, T. H. B. Gladstone, S. R. S. Godkin, N. G. Graham, R. Greenwood, D. B. Griffiths, G. H. Gunson, W. Haigh, W. J. Hanbury, B. H. Hand, J. E. Hargreaves, T. H. Hargreaves, R. D. Harland, Elizabeth M. Harper, N. Harris, H. Hassall, R. K. Hayden, Margaret I. Hendrie, Joyce M. Hierons, D. E. H. Hoffman, M. Holborow, R. F. Hollick, J. P. Hopewell, B. E. C. Hopwood, N. H. Howes, Mary W. P. Huddart, W. G. H. Hunt, J. K. Irving, R. G. Isaac, M. O. Jenks, Elizabeth M. Jenman, A. I. Joffe, D. W. Johnson, M. W. Johnson, E. N. M. Johnston, A. Jolleys, D. J. Jones, H. R. Jones, J. L. Jones, Mary P. L. Karney, G. E. Keen, D. Kennedy, M. C. S. Kennedy, P. R. N. Kerr, C. H. R. Knowles, M. Knowles, R. G. Law, Kathleen M. Lawrence, H. L. Leaming, T. Lee, M. Lewis, T. O. Lewis, J. Lister, P. Lister, B. R. Little, R. Loveless, R. S. McDonald, K. B. McKay, W. Mackenzie, J. D. M. McLaren, P. G. Mann, M. G. Marks, Helen G. T. Maycock, Helen M. Mellor, Alexa Mitchell, Elizabeth R. Mitchell, J. N. Mitchinson, D. A. Mitchison, D. B. Moffat, H. E. A. Moody, L. J. Morgan, J. L. Morris, N. F. Morris, P. G. Mundy, Diana J. Myott, F. A. Nash, W. J. Naunton, Edith F. Newling, J. A. Nissim, J. K. Ogden, A. J. P. Oldham, J. K. O'Malley, F. T. Page, G. B. Parker, T. M. Parry, J. R. Partridge, B. D. Patel, J. C. R. Payne, P. R. Peters, M. G. Philpott, H. L. Pierce, D. R. L. M. Poirier, J. McE. Potter, K. C. Precope, J. R. Preston, M. Prosser, S. G. W. Rainsbury, D. H. Randall, R. L. Ray, T. L. Reeves, P. P. Reichenheim, J. H. Rey, J. K. Roberts, M. J. Robertson, G. H. A. Robinson, P. J. L. Roche, P. M. Rooze, W. D. Rowe, P. H. Schurr, S. J. Smith, T. B. Smith, W. K. Somerton, E. R. O. E. Spearing, Joan E. Spencer, A. Stafford Steen, F. M. G. Stammers, S. Stecher, D. A. Stickland, D. F. Street, A. L. G. Stuart, W. F. Styles, I. H. Syed, Hedwig Symonds, Zahra Taki, R. G. Tasker, G. Taylor, H. N. Taylor, N. B. Thadani, D. W. Thomas, J. B. Thomas, K. D. W. Thomas, P. J. B. Thomas, P. A. Thorpe, J. M. Titcombe, A. K. Toufeeq, J. D. Trethowan, M. Trosser, H. Turner, W. J. Turney, S. M. Vine, H. N. Waller, D. Waxman, B. J. Webb, J. D. Whitby, D. Whitfield, W. N. Wild, J. R. F. Williams, Marion Williams, T. B. Willis, B. H. Wilshere, J. N. Wilson, J. R. Wilson, K. L. Wilson, R. E. Wilson, O. H. Wolff, C. R. Woodard, R. G. Woodsend, Barbara D. Wright, J. A. H. Wylie, H. Zaloudek, C. J. Zerny, J. Zimmermann.

Diplomas in Medical Radiology were granted, jointly with the Royal College of Surgeons of England, to the five persons whose names were published in the *Journal* of March 20 (p. 368), as were the names of the eleven persons who were granted Diplomas in Ophthalmic Medicine and Surgery. Diplomas in Child Health were granted to the fifteen persons whose names were published in the report of the meeting of the Royal College of Surgeons of England in the *Journal* of April 24 (p. 524).

ROYAL COLLEGE OF OBSTETRICIANS AND GYNAECOLOGISTS

At a meeting of Council held on May 1, with the President, Sir W. Fletcher Shaw, in the chair, the Honorary Fellowship of the College was conferred on Sir Wilson Jameson, Chief Medical Officer of the Ministry of Health, in recognition of his services to obstetrics and gynaecology.

Gladys Hill and N. L. White have been admitted to the Fellowship, and the following were admitted to the Membership: Janet E. Bottomley, Phillis Dingle, Marjorie O. Dunster, L. Resnick, G. S. Sturtridge, K. G. P. Worner.

At the annual general meeting of the College, held on May 1, with the President in the chair, the following were elected to Council in place of those retiring by statutory rotation: *Representatives of the Fellows*: A. J. McNair, A. A. Gemmell, J. Chassar Moir, R. H. J. M. Corbet. *Representatives of the Members*: B. L. Jeaffreson, J. S. Quin.

Medico-Legal

CRIMINAL NEGLIGENCE: A CONVICTION QUASHED

A recent judgment of the Privy Council re-emphasized the distinction between the negligence which gives grounds for a civil action and that which exposes a person to criminal penalty. A West African medical practitioner, Dr. J. O. Akerele, was touring the Owerri Province of Nigeria and treated at two villages a large number of patients for yaws. He used injections of sobita, a preparation of bismuth sodium tartrate extensively used for this purpose. Of the 57 children he treated at one of the villages 10 died.

The Trial in West Africa

He was charged before an assistant judge with the manslaughter of one of the children, Kalu Ibe; with reckless and negligent acts in giving medical treatment to the child in a manner so rash or negligent as to endanger human life or to be likely to cause harm; and with reckless and negligent acts in dispensing or administering medicine or poisonous or dangerous matter to the child. Another medical practitioner who had treated the child ten days after the inoculation, and a district mission worker, gave evidence that the boy had suffered from stomatitis induced by bismuth poisoning, and this medical witness and another doctor inferred that the death was due to an overdose. Dr. Akerele maintained in his defence that Kalu Ibe had been peculiarly susceptible to a dose which would have been harmless to a normal child. He said he had dissolved the powder in sterile water and carried a 20-oz. bottle made up for the day's work. After the illness had shown itself he returned, provided mouth-wash and milk at his own expense, and tried unsuccessfully to enlist an assistant to continue the treatment.

The judge found that the doctor had negligently prepared too strong a mixture and that his negligence was criminal. Summing up, he said that the dissolving of sobita in water to a certain strength evidently involved no particular technical finesse, but the accused in giving these injections had managed to make at least 11 persons gravely ill with the most revolting symptoms. One would have thought (he said) that in the circumstances he would at least make as certain as was humanly possible that he injected the correct dose. The judge concluded that the doctor had acted with a degree of negligence that could only be described as criminal. He found him guilty of manslaughter and sentenced him to three years' hard labour; not guilty of the second charge (negligent medical treatment), but guilty of the third charge (negligence in administering medicine), for which he imposed a fine of £100. The West African Court of Appeal quashed the sentence on the third charge, affirmed the conviction for manslaughter, but varied the sentence to a fine of £500. The Board of Examiners for Nigeria erased the doctor's name from the local medical register.

Appeal to Privy Council

Dr. Akerele appealed to the Privy Council, and the Board found unanimously that he had been wrongly convicted.[1] They referred to the leading cases on criminal negligence, and emphasized that criminal negligence must be gross, and that neither a jury nor a court can transform negligence of a lesser degree into gross negligence merely by giving it that appellation. There is a difference in kind as well as of degree. In *R.* v. *Crick*,[2] Chief Baron Pollock said it would be most fatal to the efficiency of the medical profession if no one could administer medicine without a halter round his neck; and the Board stressed the care which should be taken before imputing criminal negligence to a professional man acting in the course of his profession. They regarded it as unfortunate that the two doctors called for the Crown were accustomed to use N.A.B. and had little, if any, practical experience of sobita, with the result that the court had no evidence on the excess of strength in the mixture which would produce the observed consequences in a normal patient, nor of how widespread among the medical profession is or ought to be the knowledge of the danger of an overdose. The known margin of error was nowhere referred to.

The only negligence on which the Crown could rely was the single act of dissolving the powder in water. It was immaterial that the symptoms were revolting and that many people were made ill; the act had already taken place and its consequences could not add to its criminality. The negligence to be imputed depended upon the probable, not the actual, result. The judge had nowhere stated in what he found the negligence to consist unless in its fatal consequences in so many persons. He had imputed to the accused repeated acts of negligence as if the doctor were to be blamed for want of care in each injection instead of for the one act of carelessness in preparing the mixture. The Board could not accept the view that criminal negligence is proved merely because a number of persons have been made gravely ill and a high degree of care has not been exercised. In their view the distinction between civil and criminal liability had been inexactly drawn, the ground of the criminal charge ill defined, and the instances of negligence increased from one to many by imputing a fresh act of negligence in the case of each death. They therefore advised the King to allow the appeal and quash the conviction.

The scanty records of criminal prosecution of doctors for negligence show how rare criminal negligence is in medical practice, and how grave a degree of recklessness has to be committed to bring a doctor within reach of the law.

[1] (1943) 2 All E.R.
[2] (1859) 1 F. & F. 519.

THE CHURCH CELLAR MURDER

At a meeting of the Medico-Legal Society on March 25 Dr. C. Keith Simpson gave an account of the means taken to identify the victim in what was known as the Baptist church cellar murder, for which Harry Dobkin was tried recently at the Central Criminal Court before Mr. Justice Wrottesley, and was convicted and executed. Dr. Simpson said that a woman disappeared after making an appointment to meet her husband, who was in arrears of maintenance. Fifteen months later some remains were found underneath a chapel in Kennington, on premises to which the husband had access as a fire watcher. Even after such a lapse of time it was possible to decide the cause of death, which was strangling, and eventually to identify the body, although it had been dismembered, partially burned, and eventually buried in lime under the floor of the cellar. The remains were discovered by chance during demolition work and were thought at first to be those of an air-raid victim.

Identification of Victim

When the remains were discovered, said Dr. Simpson, the head was away from the trunk, and scalp, hair, face, lower jaw, and hands and feet were missing. It was at once seen that the remains were those of a woman, for the uterus was visible, and eventually was proved to have a fibroid tumour, which turned out to be an important point in identification, for it was found that the missing woman had attended at two hospitals for an internal complaint, which was diagnosed as uterine fibroid, and had refused operation. The murderer, after having tried unavailingly to dispose of the trunk by burning, had sprinkled powdered slaked lime over it and buried it under a large slab, but the lime, instead of accelerating the destruction, had preserved the soft parts from decomposition. The stature of the victim was estimated by two methods. The body was reassembled, and after making due allowance for missing bones and soft tissues, the height was estimated as 5 ft. 0½ in., control estimations being made by Pearson's formulae and Rollet's tables, taking the length of the left humerus, the only long bone available. Examination was made by *x* rays of certain bones in order to determine age. The state of fusion between the plates of the vault of the skull, confirmed by the palate, suggested an age of 40 or 50, and the missing woman was said to have been 5 ft. 1 in. in height and to have been aged 47. The teeth of the upper jaw clinched the examination. A dental surgeon was found who was able to produce the most detailed record cards of the treatment he had given to the woman, and from his records a picture of the upper jaw was reconstructed, which proved to be a perfect reproduction of the jaw which was discovered. Further, a full-face portrait of the missing woman was found, and as the major part of the skull could also be reproduced full face, two *x*-ray photographs were standardized and superimposed, the negative of the skull and the positive of the portrait, and by giving them exactly the same width between the orbits, the portrait fitted the skull like a mask. In the general contours of vault, cheeks, and upper jaw, and in the width and breadth of the sockets and position and shape of the nose openings, there was no dissimilarity whatever.

The Cause of Death

It remained to determine the cause of death. When the voice box was dissected out it was found that there was some deep crimson clot penetrating between the tissues surrounding the upper horn of the right wing of the thyroid cartilage. Under the bruise lay a fracture of the horn, and it was reasonable to infer that the throat of the victim had been gripped with sufficient strength to break that small bone. It was a strictly confined injury commonly seen only in manual strangulation. Dr. Keith Simpson concluded by saying that the case illustrated in a classical manner the striking results which might follow even in the most hopeless-looking cases by the patient application of simple medico-legal principles of study.

No. 16

INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a summary of Infectious Diseases and Vital Statistics in the British Isles during the week ended April 24.

Figures of Principal Notifiable Diseases for the week and those for the corresponding week last year, for: (a) England and Wales (London included). (b) London (administrative county). (c) Scotland. (d) Eire. (e) Northern Ireland.

Figures of Births and Deaths, and of Deaths recorded under each infectious disease, are for: (a) The 126 great towns in England and Wales (including London). (b) London (administrative county). (c) The 16 principal towns in Scotland. (d) The 13 principal towns in Eire. (e) The 10 principal towns in Northern Ireland.

A dash — denotes no cases; a blank space denotes disease not notifiable or no return available.

Disease	1943					1942 (Corresponding Week)				
	(a)	(b)	(c)	(d)	(e)	(a)	(b)	(c)	(d)	(e)
Cerebrospinal fever	83	10	29	3	4	170	12	32	4	8
Deaths		—	2				1	—		
Diphtheria	710	42	181	90	33	750	47	162	41	33
Deaths	15	—	2	2	2	25	—	2	—	—
Dysentery	69	7	26		—	185	18	58		3
Deaths		—		—	—		—		—	—
Encephalitis lethargica, acute	—	—	—	—	2	3	—	—	—	—
Deaths		—	—				—	—		
Erysipelas			47	7	1			50	9	2
Deaths		—	—				—	—		
Infective enteritis or diarrhoea under 2 years				5		—			31	
Deaths	47	15	3	18	4	35	3	9	7	4
Measles	13,490	860	505	36	102	3,502	297	616	138	75
Deaths	11	1	2	1	1	2	—	1	1	1
Ophthalmia neonatorum	101	7	24	—	—	83	5	17	—	—
Deaths										
Paratyphoid fever	5	—	2		1	18	—	2		—
Deaths	—	—	—		—	—	—	—		—
Pneumonia, influenzal*	866	67	13	3	17	951	56	11	9	—
Deaths (from influenza)	30	1	1	1	1	20	1	—	—	—
Pneumonia, primary		—	212	20			—	296	19	
Deaths		—		6	8		—		16	10
Polio-encephalitis, acute	—	—				4	1			
Deaths		—					—			
Poliomyelitis, acute	7	—	2	4	—	2	—	1	1	—
Deaths		—					—			
Puerperal fever		—	11	2	—	—	—	16	—	—
Deaths		—					—			
Puerperal pyrexia	156	8	9		—	186	15	16		1
Deaths						—	—			
Relapsing fever	—	—			—	—	—			—
Deaths		—					—			
Scarlet fever	1,770	164	211	39	57	1,031	46	172	37	27
Deaths	1	—	—	1	1	—	—	—	—	—
Small-pox	—	—	—		—	—	—	—		—
Deaths	—	—	—	—	—	—	—	—	—	—
Typhoid fever†	3	—	6	8	—	8	—	3	10	—
Deaths	—	—	—	—	—	1	—	—	—	—
Typhus fever	—	—	—	—	—	—	—	—	—	—
Deaths			—	—	—			—	—	—
Whooping-cough	1,827	153	152	28	36	1,672	202	100	55	12
Deaths	9	1	5	3	—	15	6	1	3	—
Deaths (0–1 year)	371	53	64	36	25	352	37	74	38	18
Infant mortality rate (per 1,000 live births)										
Deaths (excluding still-births)	4,401	631	610	193	157	4,565	650	637	217	130
Annual death rate (per 1,000 persons living)			13·7	12·7	‡			14·3	14·5	‡
Live births	6,105	724	913	372	296	6,089	592	951	316	294
Annual rate per 1,000 persons living			18·6	24·5	‡			19·7	21·1	‡
Stillbirths	228	22	35			258	33	46		
Rate per 1,000 total births (including stillborn)			37					41		

* Includes primary form for England and Wales, London (administrative county), and Northern Ireland.

† Includes paratyphoid A and B for Northern Ireland.

‡ Owing to evacuation schemes and other movements of population, birth and death rates for Northern Ireland are no longer available.

EPIDEMIOLOGICAL NOTES

Discussion of Table

In *England and Wales* the incidence of infectious diseases continues to fall—measles by 3,242, scarlet fever 288, acute pneumonia 273, whooping-cough 117, dysentery 38, cerebrospinal fever 20, and diphtheria by 13—in the week under review.

The sharpest drops in the notifications of measles were: Hertfordshire 301, Glamorganshire 282, Middlesex 240, Surrey 183, Warwickshire 182, London 181, Staffordshire 180, Lancashire 145, Carmarthen 133, Durham 130, Yorks West Riding 124, Bedfordshire 122, Kent 113, Yorks North Riding 103. A few counties reported a higher incidence: Cornwall by 86, Leicestershire by 79, Derbyshire by 53.

Although there were 117 fewer cases of whooping-cough, fluctuations among the counties were considerable. The combined totals of the divisions of London, South-East, West Midlands, North Midlands, and Northern Counties showed an increase of 108. The only large variations in the county totals were decreases in Lancashire of 59, and in Cheshire of 46.

Scarlet fever and acute pneumonia declined in incidence throughout the country, whilst the trend of diphtheria underwent little change.

The 69 notified cases of dysentery were distributed over nineteen counties, the largest returns being those of Surrey 9 and Gloucestershire 9.

In *Scotland* the principal decreases were: measles by 182, whooping-cough 125, scarlet fever 49, acute pneumonia 38, and dysentery by 19. In Glasgow the notifications of measles and whooping-cough were 109 and 95 fewer than in the preceding week, and in Edinburgh there were 22 and 23 fewer cases respectively of measles and scarlet fever.

In *Northern Ireland* the slight rise in the measles figure was due to the experience of the city of Belfast, where all the cases notified were.

Tuberculosis

The numbers of cases of tuberculosis notified in England and Wales together, and in Wales alone, in 1938, 1941, and 1942, and the percentage increases or decreases in age groups, are as follows:

Number of Cases Notified

Year	England and Wales			Wales		
	Pulmonary	Non-pulmonary	Total	Pulmonary	Non-pulmonary	Total
1938	43 205	14,888	58,093	3,096	1,129	4,225
1941	46,256	13,720	59,976	3,286	1,107	4,393
1942	46,734	13,998	60,732	3,262	1,025	4,287

Notifications in Age Groups

England and Wales

Ages:	0–	1–	5–	10–	15–	20–	25–	35–	45–	55–	65–	Total
1938	439	2,852	4,434	3,543	6,823	7,715	12,417	8,022	5,997	4,092	1,759	58,093
1941	452	2,921	3,553	2,918	7,132	8,197	13,240	8,932	6,301	4,378	1,952	59,976
1942	372	2,783	3,729	3,032	7,129	9,133	12,944	9,179	6,160	4,336	1,935	60,732
% increase or decrease in 1942 compared with:												
1938	−15	−24	−16	−14	+4	+18	+4	+14	+3	+6	+10	+5
1941	−18	− 5	+ 5	+ 4	—	+11	−2	+3	−2	−1	− 1	+1

Wales

1938	20	208	254	293	581	631	903	535	406	266	128	4,225
1941	18	205	259	253	645	684	1,006	625	357	237	104	4,393
1942	19	186	248	206	589	706	950	629	355	289	110	4,287
% increase or decrease in 1942 compared with:												
1938	−5	−10	−2	−30	+1	+12	+5	+18	−12	+ 9	−14	+1
1941	+6	− 9	−4	−18	−9	+ 3	−5	+ 1	—	+22	+ 6	−2

The increased incidence in 1941 and 1942 affected the adult population, while the notifications at ages under 15 were less than in 1938. In England and Wales an increase occurred in each age group above age 15 during 1941 compared with 1938. The largest increases were in the age groups 20–25 and 35–45, and in 1942 a further increase was recorded in these age groups while a slight decline occurred at all other ages, but the incidence remained during 1942 above the 1938 level for all ages over 15.

The Week Ending May 1

The notifications of infectious diseases in England and Wales during the week included the following: scarlet fever 1,683, whooping-cough 1,640, diphtheria 667, measles 13,104, acute pneumonia 906, cerebrospinal fever 86, dysentery 135, paratyphoid 2, typhoid 16.

Medical News

The annual meeting of the Royal Medical Benevolent Fund will be held at 3 p.m. on Tuesday, May 25, at the Royal Society of Medicine, 1, Wimpole Street, W., when the financial statement for the year 1942 will be presented and the officers, committee, and honorary auditors elected for the current year.

A meeting of the Tuberculosis Association will be held at Manson House, 26, Portland Place, W., on Friday, May 21, at 4 p.m., for a discussion on "Wartime Diet and the Tuberculous Patient."

A lecture arranged by the British Institute of Philosophy, entitled "Freewill, Determinism, and Rational Choice," will be given by Mr. D. G. C. Macnabb of Oxford at University Hall, 14, Gordon Square, W.C.1, on May 21 at 5 p.m. Cards of admission may be obtained free of charge from the Director of Studies at University Hall.

A meeting of the Council of the Medical Superintendents' Society will be held at the Midland Hotel, Derby, on Sunday, May 23, at 2 p.m.

The London Association of the Medical Women's Federation will hold a clinical meeting at the Royal Free Hospital on Saturday, May 22, when diseases of children will be demonstrated.

A week-end course on factory medical services and industrial diseases will be held in the medical school of Manchester University on May 22 and 23. "Nutrition of the Industrial Worker in Wartime," speaker Dr. E. H. Capel, is the subject for the Saturday afternoon. On Sunday morning Prof. Daniel Dougal and Dr. Joan Harwood will speak on "Gynaecological Conditions affecting Industry," and in the afternoon Mr. W. Sayle Creer will discuss "Food Faults related to Form and Function." The fee for the course is one guinea.

At a meeting held in London on May 4, sixty practitioners being present with Lord Horder in the chair, the British Association of Physical Medicine was formally constituted. The members of the inquiry committee were elected as the nucleus of the council of the association. Practitioners interested in the new association are invited to nominate a name or names of registered medical men or women to serve as additional members of the council. These names should be sent to the acting Secretary, Sir Frank Fox, 106, Finchley Road, N.W.3, before May 20. They will be submitted to a ballot at the next meeting of the association, to be held at 11, Chandos Street, W., at 2.30 p.m. on Tuesday, May 25.

A Women's Advisory Council of the Nuffield Provincial Hospitals Trust has been formed, the members of which are associated with almoning, domestic science, the general public, health visiting and district nursing, industrial psychology, local authorities, mental health, nutrition, recruitment, rehabilitation, and social service. The terms of reference include assisting the Trustees in the organization and development of medical nursing, welfare and health services, especially in relation to the co-ordination of hospital services, of social medicine, and of such other matters as will further the interests of women. Mrs. Montagu Norman is to be chairman and Miss Frances Goodall, S.R.N., General Secretary of the Royal College of Nursing, honorary secretary.

The Capetown Postgraduate Medical Association, whose headquarters are at the University of Capetown Medical School, Mowbray, C.P., South Africa, now publishes ten times a year a journal bearing the title *Clinical Proceedings* (price 2s. 6d.).

On May 8 Dr. F. Parkes Weber celebrated a double anniversary: the half-centenary of his election to the honorary staff of the German Hospital, of which he is still senior physician, and his eightieth birthday.

On May 3 President Roosevelt appointed Brigadier-General Norman T. Kirk to be Surgeon-General of the U.S. Army in succession to Major-General James C. Magee.

Over eighty local authorities have passed resolutions supporting proposals for the prevention of smoke in post-war building that have been made by the National Smoke Abatement Society (94, Manor Green Road, Epsom, Surrey). The list, published in the current number of *Smokeless Air*, includes most of the larger cities and boroughs and represents over ten millions of the urban population.

A hundred men are now carrying out the most intensive mosquito control and eradication campaign ever launched by the Brisbane City Council. They are spraying creeks and watercourses, draining low land, and seeking particularly the haunts of mosquitoes carrying dengue.

The Marie Denny Memorial Trustees have made a gift of the value of £37,000 to the Queen Elizabeth Hospital for Children, Hackney Road.

Letters, Notes, and Answers

All communications in regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1.

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B.M.A. SCOTTISH OFFICE: 7, Drumsheugh Gardens, Edinburgh.

ANY QUESTIONS?

Precautions in Haemophilia

Q.—*What is the modern treatment for haemophilia? What precautions should a general practitioner take before performing a minor operation or advising, for example, tooth extraction?*

A.—There is no generally accepted maintenance treatment for haemophilia. Claims for the value of extracts of egg white or plant extracts must be regarded as unconfirmed. The patient's blood group should be determined before operation, and if he is anaemic a pint or more of fresh blood should be given shortly before or during operation. After operation it is best to wait and see, for bleeding is neither inevitable nor predictable. If the patient bleeds after operation, apply Russell-viper venom (Boots or B.W. & Co.) or thrombin (Maw) locally, taking care to avoid undue pressure or trauma, and transfuse as often as necessary to relieve anaemia and reduce the coagulation time.

Position of the Grey Matter

Q.—*Why is the grey matter on the outside of the cerebrum and on the inside in the spinal cord?*

A.—In primitive animals the grey matter of the central nervous system was inside and the white matter outside, as it is in the spinal cord of man to-day. Such an arrangement, however, did not allow for an expansion of the nervous system to any great degree. Attempts have been made during the course of evolution to expand the central nervous system by increasing the amount of central grey matter—e.g., the corpus striatum in birds. But there is clearly a limit to expansion by this means. It is for this reason that the grey matter is developed on the outside of the cerebrum. In order to reduce the length and volume of nerve fibres to a minimum, the cerebral cortex, by virtue of the vast number of connexions which it makes, must remain a thin layer. In order that the cortex may nevertheless expand, convolutions have appeared and thus made it possible for the grey matter to expand to an enormous extent without a corresponding increase in the volume of the brain.

Counting Spermatozoa

Q.—*I have been wanting to perform my own spermatozoa counts when investigating cases of sterility. I have been unable to find among my books of reference any satisfactory account of the manner in which one should conduct an examination of a condom specimen. Would you be kind enough to give me the name of a book which gives the details of this examination?*

A.—The most complete account of modern semen analysis will be found in *Human Sterility and Impaired Fertility*, by Lane-Roberts, Sharman, Kenneth Walker, and Wiesner. Some of the methods employed require special equipment, but the counting of spermatozoa and the examination of their morphology can be carried out without this.

Lingual Ulcers

Q.—*I have a female patient, aged 33 years, who has been troubled with ulcers on the tongue and on the mucous membrane of the mouth. She has had these for four years. They vary from very small ones about 1/8 of an inch in diameter to deep, ugly, excavating ones half an inch in diameter. They are very painful. She has four artificial teeth and wears a small dental plate. The teeth have been checked by a dentist and by x-ray examination. The Wassermann reaction is negative. Urine is normal. In fact everything appears normal. I have given her the following treatment: (1) small doses of novarsenobillon intravenously; (2) courses of sulphapyridine and sulphanilamide; (3) courses of stock staph. and strep. vaccine;*

(4) courses of calcium both intravenously and intramuscularly; (5) the usual local treatment. All have failed miserably. Any suggestions will be welcomed.

A.—The diagnosis of persistent ulcers of the tongue in the absence of evidence of neoplasm or syphilis is often difficult. In the present case the duration excludes the former and the Wassermann reaction the latter. There are three possibilities, however, which require further investigation—tuberculosis, vitamin deficiency, and electrolytic effect from dissimilar metals in the denture and any tooth stoppings. Tuberculosis is best confirmed by biopsy, but search of the stools (or stomach washings) for the organism may be of help. The distribution of the ulcers outside as well as on the tongue would agree with a vitamin deficiency, particularly, of course, the B factors, while the dentist should be able to express an opinion as to the likelihood of electrolytic action

Immunization against Pertussis

Q.—*In your review of "Preventive Inoculation," by W. Powell Phillips and C. Woodroffe Anderson (March 20, p. 353), you say, "It is pleasing to note this advocacy of immunization against pertussis; unfortunately the recent work of Bell on the efficacy of two doses of pertussis vaccine at a month's interval instead of 4 to 5 weekly injections came too late for inclusion in the text." There seems to be a good deal of controversy about the best method to adopt. I should be greatly helped if you could kindly tell me which is the work of Bell in which I can see his conclusions on this subject.*

A.—Most of the field trials on the prophylaxis of pertussis by specific vaccines have been done in the U.S.A., where the usual practice is to give 3, 4, or 5 weekly injections of smooth-phase vaccine (10,000–20,000 million organisms per c.cm.) totalling 80,000–100,000 million organisms. To avoid frequent injections of such big doses, which for large-scale immunization introduce difficulties in both production and administration, Bell[1] carried out a well-planned and well-controlled experiment in Norfolk, Virginia, using two 1-c.cm. doses (10,000 million organisms per c.cm.) of alum-precipitated vaccine at one month's interval. Very few children had any systematic reaction. There was some local tenderness and stiffness, but if the vaccine was not injected too superficially the firm nodule which usually developed was not noticed by the parents. The results were similar to those obtained with more frequent and larger doses. In the period of 34 months after vaccination 11.2% and 9.4% of two different age groups developed pertussis, compared with 39.1% and 29.7% among control uninoculated children. As usually happens, pertussis in a vaccinated child was milder and of shorter duration than the attack in uninoculated children. Since then Kendrick[2] has carried out comparative tests with plain and alum-precipitated vaccines, giving three 10,000-million doses of each at intervals of one week between the first and second doses and four weeks between the second and third doses, and she found no significant difference in the protective value of the two vaccines.

Diphtheria toxoid and pertussis vaccine may be combined without any inhibitory effect on the immunizing property of either antigen. It must be remembered, however, that pertussis has an earlier age incidence than diphtheria; in fact, about half the deaths from pertussis occur in the first year of life, so that specific prophylaxis should be begun at three to six months of age.

LETTERS, NOTES, ETC.

The Imperial Institute's Jubilee

On May 10, 1893, one of the least publicized but most useful national institutions in Britain was opened. It was the Imperial Institute in South Kensington, and ever since Queen Victoria performed the ceremony the Institute has carried on with the work for which it was founded. Designed as a shop window and show room in which could be exhibited all the wealth of raw materials that the British Empire can provide, the Institute also had two other functions which it has admirably performed during its 50 years of life. In the laboratories a scientific staff examines samples of minerals and plant and animal products which are submitted from all over the Empire with a view to their being put to commercial use. Not long ago the Colonial Office was anxious to have an assessment made of the nutritional properties of several native foodstuffs grown in Nyasaland; the experts in South Kensington were able to provide the answer. In the plant and animal products department oils, oil seeds, gums, fibres, insecticides, and other materials from all over the Empire can be scientifically examined and their use decided; and the same type of work is being carried out on the Empire's mineral resources in the minerals department. For example, wild rubbers found in Africa are being tested for elasticity and durability—it is hoped that they may help to replace the supplies of cultivated rubber lost in Malaya. Further, the Institute sends films and lecturers to schools and discussion groups all over Britain.

[1] *Publ. Hlth. Rep.*, Wash., 1941, **56**, 1535.
[2] *Amer. J. publ. Hlth.*, 1942, **32**, 615.

Malignant Tertian Malaria

Dr. RICHARD FAWCITT (Ulverston) writes: I have read with interest an account of a case of malignant tertian malaria by Drs. W. L. Anderson and D. B. Bradshaw (April 24, p. 508). I should like to draw attention to the fact that a number of cases were treated at the Albert Dock Hospital in the summer and early autumn of 1940. These cases came from a ship travelling to England by the West African route. My son, Fl. Lieut. J. B. Fawcitt, now serving abroad, was at that time acting as house-physician at the hospital, and, by the kind permission of Sir Philip Manson-Bahr, published an article recording some of the cases in the *London Hospital Gazette* (1940, **44**, No. 1, 9) under the heading of "Subtertian Malaria at the Albert Dock Hospital." Furthermore, he recorded a case in conjunction with A. H. Walters under the title of "Acute Malignant Tertian Malaria: Treatment aided by Reconstituted Serum" in the *British Medical Journal* of Jan. 4, 1941 (p. 14). I trust that these references may be helpful.

Cramp in Arteriosclerosis: A Cure

Dr. J. W. J. WILLCOX (Glastonbury) writes: The one treatment which will cure most cases of cramp at night associated with arteriosclerosis is 3 gr. of quinine sulphate at bedtime. I have not known it fail in any case during the past 3 years, and I can produce 4 patients at the present time who find it infallible. Its use for this purpose is supported by the literature on the subject, to which I am indebted for its introduction, but I confess that my search for a cure extended over 25 years.

⁂ Under an order, which came into force on Jan. 5 last, quinine and its salts may not be prescribed except as antimalarial drugs.—ED., *B.M.J.*

Supply of Occupational Therapists

Dr. ELIZABETH CASSON writes: May I be allowed to correct an error in the article on rehabilitation in civilian hospitals (April 10, p. 454). It is stated that the sources of supply of masseuses and occupational therapists are the Chartered Society and Dorset House School respectively. "Dorset House School" should read "The Association of Occupational Therapists." The profession is young and therefore small, but it has organized itself well, and though the Dorset House School has been privileged to supply a considerable number of the personnel required for the E.M.S. hospitals, it is only one of several schools whose pupils take the Association examination.

External Application of Opium

Dr. D. PRIESTLEY SMITH (Birmingham) writes: There must be many who can confirm Dr. J. McLaren's observations (March 20, p. 370) that opium and its derivatives do take effect through the skin. It was common knowledge to our medical forefathers. Here is a striking example which anyone can verify: Dissolve two grains of morphine in a drachm of oleic acid, and, with the finger-tip, dab it lightly on the painful area in a case of herpes zoster ophthalmicus. In ten minutes you will find the patient's pain relieved and your finger-tip numb. I do not know of any other treatment that gives the patient so much relief in this condition. Those who say that opium preparations applied externally are useless state what is simply not true. The men of former generations knew better.

"Scientific" or "Unlimited" Freedom?

Dr. DONALD M. O'CONNOR (Brenchley, Kent) writes: The spiritual descendants of the courtiers of King Canute who belittle all existing public medical services and attempt to denigrate any which are suggested for the future exhibit an amazing complacency with the present state of our affairs. Their latest war-cry is a demand for "scientific freedom." During recent years a great part of the profession has exercised its scientific freedom to abstain from learning the indications for the use of sulphapyridine, and to refrain from ascertaining whether these existed in any given case, so that this potently toxic drug has been lavishly administered for any and every febrile condition, and we have arrived at a state of affairs when nearly every mother of a pyrexial child asks on one's first visit, "Aren't you going to give M & B, doctor?" Unlimited freedom, whether it can be dubbed "scientific" or not, has its dangers.

Disclaimer

Dr. EUSTACE AKWEI of Accra, Gold Coast, West Africa, wishes to state that he has no connexion of any kind with Lieut. Eustace Alar Akwei, R.A.M.C., who was referred to in the London *Evening News* of Nov. 25, 1942, under the heading "African posed as Bachelor of Surgery."

Corrigendum

In the leading article on "Medical Applications of the Cyclotron" (May 1) the identity of the Lawrence brothers was confused. The work referred to in column 1 of page 543 is that of E. O. Lawrence (Nobel prizeman) and not J. H. as mentioned in line 11. J. H. Lawrence's work is that described in the middle of column 1 of page 544.